Chiropractic Management of a 24-Year-Old Woman With Idiopathic, Intermittent Right-Sided Hemiparesthesia

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Abstract

Objective: The purpose of this case report is to describe the chiropractic management of a patient with idiopathic, intermittent right-sided hemiparesthesia.

Clinical Features: A 24-year-old woman presented with a 2-year history of intermittent idiopathic right arm paresthesia. She also had a 3-month history of intermittent idiopathic right leg/face paresthesia. These symptoms were strongest at night and caused insomnia and worsened over time. She rated her discomfort at 5/10 on a numeric scale.

Intervention and Outcome: Care included vibration stimulation therapy, spinal manipulation and cold laser therapy. She had a noticeable reduction in her paresthesia both subjectively and objectively. She showed improvement in paresthesia on the right side of her body after the first visit. The following week, after 2 visits she returned and stated that she was symptom free with 0/10 discomfort on a numeric scale.

Conclusion: This patient's symptoms of idiopathic, intermittent right-sided hemi-paresthesia seemed to improve with a short course of chiropractic care using manipulation, vibration therapy and cold laser therapy.

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Introduction

Intermittent paresthesia is described as abnormal unpleasant numbness, pins-and-needles sensation, or tingling that happens spontaneously without external

sensory stimulus.¹ These sensory experiences can be caused by a variety of neurological conditions.² Paresthesia is often associated with specific nerves and therefore is referred to as a neuropathy.¹ Neuropathies are distinguished from general paresthesia if there is dermatomal pattern to the distribution.³ If the paresthesia is directly due to specific nerve compromise, an individual will experience numbness or tingling along the specific distribution similar to carpal tunnel syndrome, herpes zoster, or a herniated disc.^{2,4} Generalized

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paresthesia does not follow a specific nerve distribution and may be due to an upper neuron lesion.³ Larger distributions of paresthesia can be signs of upper neuron compromise stemming from the spinal cord or cortical circuits.¹ There are numerous types of peripheral neuropathies with varying signs and symptoms.

General examination techniques such as sensory discrimination testing with sharp, dull, vibration, and temperature should be done to distinguish sensory neuropathy and upper neuron compromise.^{2,3,5} Performing a sensory examination will help to determine between upper and lower motor neuron lesions, with upper motor neuron lesions having spastic paralysis and a lower neuron lesion having flaccid paralysis. Quantitative sensory testing is also used as a means of sensory nerve testing; however, using quantitative sensory testing should not be the sole measure used for diagnosing pathology.^{2,8} Electromyography may be used to check for large-diameter nerve conduction problems; however, it does not differentiate between simulated sensory loss and neuropathy.^{1,9}

Medical treatment method for idiopathic sensory paresthesia depends on controlling neuropathic pain, which can be treated with antiseizure medications, antidepressants, analgesics including opiate drugs, or a spinal cord stimulator.^{2,10} There are no complementary alternative medicine therapies that have been documented as being effective for the treatment of idiopathic paresthesia. The purpose of this case report is to describe the chiropractic management of a patient with idiopathic, intermittent right-sided hemiparesthesia.

Case Report

A 24-year-old Caucasian woman with a 2-year progressive history of idiopathic hemiparesthesia sought help for her worsening condition. When asked how her symptoms started, she could not recall an exact date or cause. She had been experiencing right arm and shoulder numbness for 2 years. The pain traveled down the outside of her arm to her elbow, was intermittent in nature, and at times wakes her up at night. Three months prior, she started having similar symptoms in her right leg, where the entire leg would get a “tingling” feeling and would have periodic twitching episodes. The episodes would last only a few seconds. Around the same time, she said that the right side of her face would experience numbness along with her right arm and leg. Using a numeric scale rating discomfort from 0 to 10, she rated her symptoms at 5/10. Nothing else in

her history was pertinent to her current symptoms, and family and past medical history was unremarkable.

An examination was performed; no aberrancies were discovered either on the right or left upper extremities using sharp or dull sensation. Vibration sense was tested and was normal in both her upper and lower extremities. All reflexes were graded at a normal 2+ except for her patellar reflexes, which were 3+ bilaterally. All muscle testing was graded at 5/5. Cranial nerve examination was normal.

On the basis of principles of neuroplasticity, other testing was performed. Her pursuing mechanisms in her lateral eye movement were examined and showed a slight difference. There was no difference seen when comparing right to left bilateral eye convergence. Nonpathological saccadic eye testing was unremarkable. She was asked to repeatedly touch her nose and then the doctor’s finger, which was 1.5 feet away in all 4 quadrants of vision. She was able to accurately touch her index finger to the doctor’s finger repeatedly with her eyes closed in all quadrants except the lower right quadrant. In this quadrant, with her eyes closed, she consistently missed the doctor’s finger and was not able to properly pinpoint the placement. Rapid alternating movements of the distal upper extremity phalanges were also performed. She was asked to rhythmically and rapidly pretend like she was playing a piano with her fingers. She showed differences in the velocity of finger movement on the left side being slower and less coordinated than the right on visual inspection. Her palate was also examined, and although there was no pathologically induced palatoparesis, which is lowering of the palate, the left side of her palate started to lower slightly when compared to the right on repeated vocalizations. A functional Romberg test was also done to test her balance. While standing with her eyes closed, she had a minor sway in the anterior posterior plane. Immediately after this, she was instructed to place her head posteriorly and to the left to see if this altered her sway. She repeated this activity, putting her head posteriorly to the right, anteriorly to the left, and finally anteriorly to the right as well. While her head was posteriorly positioned to the left, her sway increased, almost causing her to lose her balance. Once this was found, she was asked to immediately perform eye fixation exercises, where she actively brought her head posteriorly to the left and then neutral as she fixated on a point in front of her. She repeated this activity 12 times and then paused to be retested. Immediately after these exercises, her sway in all planes ceased.

She had taut and tender muscular points located at C6 and 7 bilaterally, with decreased motion in all

ranges of motion. After the history, observation, and physical examination, a working diagnosis of idiopathic hemiparesthesia on the right was obtained.

During examination, different modalities were used to affect her symptoms. After she demonstrated her inability to perform accurate finger-to-nose testing on the right lower visual quadrant, we placed vibration on her right side. This was directed at her right ankle mortise joint, knee, lateral hip, wrist, elbow, and anterior shoulder area. The percussion used was a speed setting of 7 on an Erchonia (McKiney, TX) percussor instrument. This was done for a period of 1 minute per area treated, as noted above. After vibration therapy was completed, she was asked how she felt. She stated that she no longer felt any disturbances on the right side of her body.

An Erchonia cold light laser was applied to her lower right cervical spine approximately 2 inches laterally to the vertebral prominence at C7. This was done for 3 minutes at diode frequencies; of 2, 9, 13, and 528.

She said she no longer was experiencing any symptoms. She returned 2 days later and reported that her symptoms returned during the following night after treatment. They did not appear to be as noticeable to her, and she noted that there was improvement. The following week, after 2 visits using the above treatment modalities, she returned and stated that she was symptom free and had been, since her second visit. She rated her discomfort on the numeric scale as 0/10.

Discussion

Functional neurology is a practice based on principles of neuroplasticity. Neuroplasticity is the understanding that our nervous system is changing and adapting to its surroundings. It is hypothesized that if there is a lack of positive plasticity within a certain area, underdevelopment or dysfunction of the neuronal pool may develop creating aberrant output. Theoretically, based on physical findings, modalities could possibly be aimed to alter neuronal firing and create changes in response. If positive change is noted, it may indicate plasticity alterations, resulting in symptom relief.

Paresthesia stems from disruption of neuronal flow.¹ When a nerve impulse is subjected to changes in its surroundings, whether metabolic or external, changes in firing rate and frequency can occur.¹ This is then perceived in higher regions of the neuraxis as altered sensation. Specifically, an individual's parietal lobe is responsible for determining what type of sensation is

felt.¹ Patients with paresthesias usually have some type of impaired perception of cutaneous sensations.¹ This is due to the small myelinated fibers carrying these sensations being selectively affected.¹ Changes in metabolic processes within the neuronal membrane can create dysfunction of the cell body.^{11,12} A prime example of paresthesia caused by metabolic insults is seen in diabetes mellitus. This type of neuropathy may cause paresthesia-like symptoms; paresthesia's can also result from upper motor neuron destruction. In the case of multiple sclerosis an individual's neuronal sheath is damaged, causing explicit changes in the nerve's excitatory capacity.⁵⁻⁷ This can be also be perceived as a paresthesia. When upper neuronal disease, metabolic disorders, and external disruptions are ruled out, one is left with a diagnosis of idiopathic paresthesia. Because no pathology can be found in individuals with idiopathic paresthesia, we must look at the symptom as a result of dysfunction within the individual's ability to process sensation. There is a "disconnect" between an individual's ability to perceive sensation and properly process information in higher neuronal structures of the cortex.

Neuronal cells may in fact change due to external stimulus, which can create new dendritic connections between cells.¹³ There are a variety of factors attributed to proper plasticity within neurons. Structures known as *N*-methyl-D-aspartate (NMDA) glutamate receptors have been linked to proper firing and health of neurons.^{14,15} If the receptors do not properly regulate neuronal membrane function, positive change may not take place within the cell.¹⁷ Research indicates the need for NMDA glutamate receptors to become regulated within postsynaptic neuronal membranes to create proper functioning of neuronal structures.^{14,15} The receptors are responsible for long-term potentiation, creating plasticity within neuronal circuits.¹⁶ Thus, creating healthy neuronal function is dependent on properly functioning NMDA receptors. If we consider that the patient's capacity to form positive plasticity within her parietal lobe was potentially insufficient, the fact that stimulus was given to influence parietal lobe functions may explain the positive response to the modalities used in this case.

N-methyl-D-aspartate regulation is dependent on stimulation of the neuron.¹⁴ If temporary stimulation is given in areas of targeting measure to excite certain neuronal groups, NMDA receptors of the given group will begin to increase in function.¹⁴ The opposite is true of NMDA receptors that are not excited by stimulus. The receptors will not properly gate action potentials, thus creating a lack of postsynaptic connectivity.¹⁴

Thus, stimulation of neurons is critical to proper interconnectivity of neuronal tissue.¹⁶ Studies have been done to show the significance of paresthesias and the consequence of negative neuronal plasticity within the somatosensory cortex, specifically for paresthesia's associated with carpal tunnel syndrome.^{18–20}

The primary somatosensory cortex is responsible for interpreting sensations. Because our examination did not find any reason for our patient's symptoms, we are left without an identifiable cause. One explanation that could explain the significance of the pain is the lack of plasticity within her neuraxis. When areas of the cortex are not properly stimulated as explained earlier, NMDA receptors will not properly gate channels and neurons lose plastic connectivity.¹⁴

Phantom limb syndrome can be used to understand possible correlations to the symptomatology experienced in both phantom limb and idiopathic paresthesia. In phantom limb syndrome, individuals may feel certain sensations in areas that are no longer part of their body.²¹ Techniques such as mirror box therapy have been used to manipulate and change the perception an individual may feel.²² This therapy has worked for those with phantom limb.²³ Mirror therapy changes the perception an individual has of his or her limb.²⁴ This supports the theory that plastic changes occur in the individual's brain. That being said, with idiopathic paresthesia, a similar mechanism may be the cause.

On the basis of principles of neuroplasticity, which include changes in neuron connectivity, a number of therapies were used. Our patient had difficulty performing finger-to-nose exercises in the lower right quadrant. This activity primarily deals with joint position awareness and perception of one's self relative to another object. This can be broadly determined to be a parietal lobe calculation. Seeing that this is a contralateral processing sensation, it was determined that her left parietal lobe did not function as accurately as her right. Because of this, she received vibration therapy to the right side of her body. This was done to create excitation of her left somatosensory cortex and overall parietal activity. As all senses are multitargeted; the parietal lobe is the most heavily incorporated in vibration reception.²⁵ The concept was that this created temporary plasticity within her somatosensory organization.

She was also tested using a functional Romberg test, which is used to test proprioception, which is directly related to the dorsal columns. To perform the test, the eyes are open and feet together while standing, and the patient is then asked to close his or her eyes and any sway is observed; this eliminates the ability to use

vision and vestibular function, depending only on proprioception for balance. She actively placed her head in varying positions while this was done to see if the change in position created an increase in her unsteady sway. Her sway increased when her head was placed in the plane of the posterior left semicircular canal. When her head went posterior and to the left, temporary excitation of the canal should have been elicited. This would have kept her steady. This is due to vestibulospinal pathways, which create spinal stabilization when activated.²⁶ Because she had sway when her head was placed in the posterior left position, it portrays a difference in its functioning as compared to the right posterior, anterior, and left anterior canal. As a result, she was told to fixate her gaze on the doctor's shirt button while she brought her head in the posterior left position while maintaining a fixed visual. This was done to elicit excitation of the posterior left semicircular canal and therefore create temporary stimulus to this area feeding into the vestibulospinal pathway.²⁶ She was then tested after exercise and no longer had any sway during the functional Romberg.

Limitations

The cause of the patient's condition was unknown; therefore, it would be difficult to hypothesize any cause and effect. It is possible that the patient had a placebo or psychosomatic response to the treatment provided. She experienced results after the first visit, which suggests the treatment was beneficial to her in some manner. The environment of the patient could not be controlled; therefore, it is possible that her improvement could have been due to other factors. Future research is needed to specifically determine whether the use of functional neurology is effective and if it can be duplicated, and if the results are dependent on the knowledge of the provider. Better objective evaluations are needed to determine the effect on the patient's symptoms with and without these treatments.

Conclusion

The patient with hemiparesthesia reported a positive outcome to chiropractic care, which included manipulation, vibration therapy, and cold laser therapy.

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No funding sources or conflicts of interest were reported for this study.

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