CHIROPRACTIC MANAGEMENT OF A PATIENT WITH CEREBELLAR ATROPHY

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ABSTRACT

Objective: To describe the chiropractic management of a patient with cerebellar atrophy.
Clinical Presentation: A 38-year-old female sought care for cerebellar atrophy of her left cerebellar lobe. She had cognitive difficulties, nystagmus in certain head positions, vertigo, difficulty with fine movement patterns, and balance issues, all of which affected her day-to-day life.
Intervention and Outcome: She was treated with neurological exercises primarily targeting her left cerebellum as well as its neurological pathways, in conjunction with chiropractic manipulative therapy. Within 3 weeks of treatment, she saw reduction in her symptoms and improvement in her condition, which caused her to have a more positive outlook for her future.
Conclusion: For this particular patient, the use of chiropractic care and functional neurology appeared to help with her signs and symptoms. Her response to treatment plateaued after 3 months of treatment as she later began to miss appointments due to external stressors. (Chiropr J Australia 20178;45:175-183)

Key Indexing Terms: Chiropractic Manipulation; Cerebellar Atrophy; Chiropractic Adjustment; Functional Neurology

INTRODUCTION

The cerebellum is a region of the brain associated with fine control and coordination of both simple and complex movements. Its primary functions include balance and fine motor skills but it may also be involved in regulating fear and pleasure responses as well as some cognitive functions such as attention and language. (1) Cerebellar atrophy is a process where neurons in the cerebellum that are responsible for controlling balance, co-ordination and movement deteriorate and die. (1)

One possible cause of cerebellar degeneration is traumatic brain injury (TBI). (2,3) Some of the components of TBI, including ataxia, postural instability, tremor, impairments in balance and fine motor skills, and cognitive deficits, may be attributed in part to cerebellar damage. (4,5) Another possible cause of cerebellar disease is inherited genetic mutations that alter the normal production of specific proteins necessary for the survival of neurons (5), such as Frederick's Ataxia, or chronic alcoholism. Direct cerebellar injury is much less common than supratentorial trauma, and radiologic studies of TBI confirm that the cerebellum is often affected even when the initial injury does not directly involve this structure. (6) Several pathophysiologic mechanisms mediating TBI-induced cerebellar damage have also been described

following direct or indirect injury: excitotoxicity leading to Purkinje cell loss, an evolving TIA, and a distinct spatial pattern of microglial activation coincident with Purkinje cell loss. (7) The most definitive symptoms of cerebellar atrophy include slow, unsteady and jerky movement of the arms or legs, slowed and slurred speech, nystagmus and wide-based, unsteady, lurching walk, which is often accompanied by a back and forth tremor in the trunk of the body. (1,4)

Two types of cerebellar syndromes exist, midline and hemispheric. (5) Midline cerebellar syndromes involve a patient's balance, titubation, as well as truncal ataxia. It may also affect eye movements, causing nystagmus, ocular dysmetria and poor smooth pursuits. Hemispheric cerebellar syndromes include presentations such as incoordination of the limbs, dysmetria, dysdiadochokinesis, and intentional as well as kinetic tremors. Speech may also be affected, causing dysarthria, scanning speech or irregular emphasis on syllables. (5,8)

The diagnosis of cerebellar disorder is often made by a neurologist, and in most cases is straightforward due to the high specificity of the signs described above. Brain imaging should be performed to properly diagnose the condition. Open magnetic resonance imaging (MRI) or low-field MRIs are not recommended. MRI indicates shrinking of all or part of the cerebellum along with possible shrinking of the brainstem. (5,8)

A possible intervention for this kind of cerebellar disorder is the application of therapeutic neuroplasticity. Plasticity involves an object's ability to undergo change. Neuroplasticity is how the nervous system, which has limited regenerative capabilities, is able to adapt to an ever-changing world. The concept is that the nervous system can change its connections to create or enhance pathways necessary to the organism. The more that a pathway is used, the more connections that are made, and therefore the easier it is for the body to activate that pathway. (9)

Plasticity has been theorized for over 100 years but has only recently been regarded as a possible means for therapy. Functional neurology is a form of manual medicine using specific joint manipulations, muscle afferentation, and vestibular and proprioceptive rehabilitation to increase stimulation to specific neural pathways and specific areas of the brain to enhance neuroplasticity within these regions. This neuroplasticity can possibly help to bypass damaged areas of the brain and allow pathways to be reformed that may have been damaged due to stroke or have atrophied due to disuse or developmental delays. (9-15)

CASE REPORT

A 32-year-old female chiropractic student sought chiropractic care for cognitive difficulties, diplopia, and nystagmus, loss of balance, vertigo and lack of coordination. Symptoms started 3 years ago following a wakeboarding accident where she fell on the board sideways and hit her head. She does not remember which side was hit. She said she does not remember what happened right after her injury and experienced a loss of

consciousness immediately following the incident. An MRI at the Mayo Clinic revealed degeneration of her left cerebellar lobe. Her MRI report noted that there was stable moderate left cerebellar atrophy with no other remarkable findings. (Fig 1 & Fig 2).

Three years following the incident she sought chiropractic care, stating she had a diagnosis of left-sided cerebellar atrophy. At first, she was tested for cranial nerve functioning, musculoskeletal, sensory and reflex examination, brain lesion as well as cerebellar orthopedic tests.

Reflexes on her left-sided extremities were all +1, except her patellar reflex which was rated +4. All right-sided extremities were +2. Muscle testing on her left side was 3/5 on both extremities, 4/5 on right-side extremities. Cranial nerve exam revealed diplopia when performing a field gaze examination and also during convergence testing. Her cervical and lumbar orthopedic testing was unremarkable. Marching in place with eyes closed revealed an initial sway of backward-left to the point of falling unless support was provided. Her gait was slow and arm swing decreased, with the left arm swing slower than right. There was also a leftward sway and drift when combined with a complex task. Leftward saccades revealed a hypermetria usually including 2 attempts or more to hit the target. Vertical saccades revealed hypermetria on lower targets with misses occurring inferior and to the left. Smooth pursuits reproduced the patient's diplopia. Static finger-nose testing was unremarkable; however, when the target was in motion during kinetic testing she often missed the target with her finger. She stated she felt fatigued after 10-15 seconds and breaks often had to be taken to complete the testing. Piano test revealed uncoordinated motion in her fingers bilaterally with decreased speed noted in the left hand.

Based on the above, a diagnosis of left cerebellar lobe atrophy was confirmed.

Improvements were seen in her condition after 3 weeks of manipulative therapy and prescribed exercises. Improvement was shown based on her own subjective report on her condition as well as objective findings by the intern and doctor of improvements in her ability to perform the complex tasks and eye movements. She originally described fatigue in her neck when performing the exercises at home. She described the fatigue as tightness in her back of the neck area that she insisted was not physical but more mental in nature. After about 3 weeks of care she was showing improvement in her symptoms, especially in balance and fine movement of extremities. She stated she could play the guitar much better than when her symptoms first started. She also had slurred speech less often, as well as improvements in balance bilaterally. As she reported earlier, she used to get headaches whenever she performed a mentally straining activity or when she was under mental stress from school, but had also mentioned beginning to get the headaches since she started school about 6 months prior. The headaches were absent for about a month following treatment; however, these returned as school began to be more stressful again and she reported she was unable to perform the exercises without pain like she was able to prior. Due to this, we limited the therapies to a tolerable limit in order to not overtax her already strained neurological system. After the headaches abated, her condition was slightly worse than

earlier and her treatment was discontinued. Due to the stall in recovery she dropped out of care to seek other alternatives.

After a 3-year break from our care she returned, stating that she had sought care with other chiropractors to no avail. She had gained minor improvements but always plateaued fairly quickly. These doctors did vestibular rehabilitation to stimulate the brain as well as upper cervical adjusting. These activities often exceeded her metabolic capacity and would make her feel sick.

Her blood pressure was taken on both arms, showing a 20-point systolic and a 10-point diastolic increase in her right arm versus her left. She had normal sensation and +5/5 muscle strength. However, she had +4/5 in her left upper extremity that caused her body to jump. Muscle strength testing on her left thumb extensor was +4/5 vs the right +5/5. She performed the Rhomberg test and could barely keep her balance with her eyes open. When she closed her eyes she immediately fell to her right side.

The left-sided hyperreflexia suggested an upper motor neuron lesion in her left cerebellum; however, a rightward sway on a Rhomberg test suggests a right cerebellar lesion. As well, the reduced thumb extensor strength and increased blood pressure on the left side showed an increase in sympathetic tone on the left side. This is associated with decreased pontomedulary reticular formation (PMRF) firing, which in turn is associated with a reduced firing of the ipsilateral cortex and contralateral cerebellum.

We started treatment by trying to activate the left cerebellum, the original hard lesion, by performing left-sided complex motions in the upper extremity, as well as adjusting the left extremity to send proprioceptive input to the left cerebellum. We also counseled her on proper breathing mechanics and adjusted her ribs to increase airflow. While these therapies seemed to help her balance slightly, the other problems that she was facing such as fluency and brain fog failed to respond. After seeing this, more right cerebellar and left cortex stimulation was tried. The same complex movement exercises were performed on her right side. She then performed mental math but wrote out the numbers on her right leg with her right hand and having her picture the numbers as she wrote them. Then she did right spins while keeping an eye on a target to stabilize her vision while using her vestibular system to strengthen the right cerebellum and left cortex. She was given these exercises to do at home. She was told to play her guitar but switch off between left and right to create general stimulation.

On her next exam day she was 2+ on all of her reflexes and all of her sensation and muscle strength tests were normal. She still had a positive Rhomberg's test with a rightward sway but even with her eyes closed she did not lose her balance. The thumb extensor strength on the left was still slightly reduced but has increased since she began her treatment. She has also stated that she has been able to go out dancing and has started to be able to play her guitar for much longer periods of time.

DISCUSSION

The use of neuroplasticity as a therapeutic intervention is a relatively new field in mainstream healthcare. There have been many publications recently addressing the use of somatic sensation to activate neural pathways and therapeutically create a neuroplastic response. (13-15) Neuroplasticity in the clinical setting is achieved through various modalities and exercises specifically designed to target neural pathways that because of a person's lifestyle or possible disabilities are not targeted in their everyday life. The concept is based on the understanding that soft lesions are cortical deficits due to developmental delays, environmental stressors, or aberrant biomechanical movements that lead to decreased output of brain activity in a particular region or regions. These soft or physiologic lesions differ from ablative lesions in that they are not permanent damage to the tissue itself. The use of this concept can be transferred to the treatment of ablative lesions by using neuroplasticity to shunt the pathways around the ablative lesion, allowing new connections to be formed through non-damaged portions of the neural pathway.

The neurologic rehabilitation utilized in this patient was first to involve simpler tasks along with passive tasks and modalities. This would increase in difficulty and complexity as she showed improvement. The initial exercise given to the patient was a complex figure-8 movement to be performed using 1 joint region, in this case her left wrist, to stimulate the left cerebellar lobe in conjunction with the thalamocortical projections to the prefrontal cortex in order to carry out the planning of, and the smooth control of the specific complex movement. The task would increase in complexity as improvements were noted in speed and smooth control by adding another joint, such as the elbow and/or shoulder, to recruit more mechanoreception along with requiring more cortical activation to plan and carry out the task. These prescribed exercises were to be performed in a safe environment for at least 10-15 minutes a day unless she became mentally fatigued and exceeded her metabolic capacity. At her twice-per-week office visits, these exercises were performed to monitor progress and were given in conjunction with chiropractic adjustments on her left side.

Following these exercises, she was next given more complex exercises such as Dix-Hallpike and Epley's. Dix-Hallpike followed by Epley's is not a definitive treatment for discharged otoliths causing nystagmus but has been shown to help. (9,16) The treatment approach here was that her nystagmus was independent of her primary diagnosis and could be treated independently. The maneuver was performed twice on different but consecutive visits but due to no change in her nystagmus further therapy to specifically treat her nystagmus was discontinued.

She was then given balance exercises for her left leg, which included standing on 1 leg initially using a support and later after a few days she felt comfortable balancing without support from an intern or other aid. This was given and expected since the cerebellum is important for making postural adjustments to maintain balance. Through its input from vestibular receptors and proprioceptors, it modulates commands to motor neurons to compensate for shifts in body position or changes in load upon muscles. (15)

She was given pursuits to a moving target with her head neutral and stationary, as well as exercises with the target stationary with the head moving horizontally left and right. Vertical and horizontal saccades were performed with her head originally placed in the neutral position with her being asked to "quickly move eyes to the target as it flashes." This was performed with the intern's thumbs being placed laterally within the patient's visual field, and moving a thumb would be the patient's indication to saccade to the target, hold the eye position, and then return to eyes neutral when instructed. The patient was also given the task of holding both of her arms out in front of her at shoulder level with her thumbs together and point upward. The patient was asked to stare at her thumbs and rotate her body, head and shoulders to the left while maintain focus on her thumbs, then rotate back to neutral and repeat. This was to be performed 30sec at a time with 30sec of rest for 2 minutes. The purpose of these exercises was to attempt to stimulate the cerebellar pathway, which fired to the left cerebellar lobe specifically to stop further atrophy of her cerebellar fibers and cells as well as for creating neuroplasticity to change the neural pathway and cells of the central nervous system to compensate for the atrophied cells. (9) She was also given chiropractic adjustments during her visits at the clinic; these were mostly focused on the left-sided segmental dysfunction in her cervical spine at vertebrae levels 2 and 6. We used a supine diversified technique.

While the use of functional neurology and therapeutic neuroplasticity is a relatively new and constantly evolving field within the chiropractic and neurological profession,we do know that certain movements and exercises can stimulate parts of the brain.(13-18) Our patient did see some overall benefits and improvements with care. Following in the footsteps of many neurologists and chiropractic neurologists in recent years who have demonstrated the effects of specific spinal manipulation, and exercise therapeutic modalities in creating neuroplastic changes and functional improvements in patient response we feel the field is growing and changing in terms of neurological advancements and treatments. (14,15,17,18)

Limitations

The patient became discouraged from seeking further care when her progress stalled. It is plausible that placebo or her psychosocial responses were responsible for the improvement in her condition. There is also no way of confirming if the patient did perform the exercises at home or if the exercises were performed correctly. Being unable to control the patient's environment could also alter the outcome of her progress.

CONCLUSION

This patient had neurological deficits and though they may have originally been because of the ablative lesion in her left cerebellum, her symptoms changed either because of disease progression or because of the treatments themselves. She may have already had a right neurological deficiency before the accident that may have worsened due to the neurological damage. A midline cerebellar stroke could have had

crossover that was not viewable on MRI. Another possibility is that the patient was being overstimulated by directly activating the damaged cerebellum. It is possible that the right-sided treatments were causing minor stimulation to the whole brain, which allowed the damaged pathways to reform enough to create neuroplasticity. It is impossible to know without better imaging and even then an explanation could elude us but this has shown that even though a hard lesion is present it may not be the entire cause of the symptomology. More research needs to be conducted to see if there are more patients similar to this and to determine the prevalence of the condition.

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