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CASE REPORT

Upper Cervical Chiropractic Management of a Patient with Parkinson's Disease: A Case Report

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ABSTRACT

Objective: To discuss the use of upper cervical chiropractic management in managing a single patient with Parkinson's disease and to describe the clinical picture of the disease.

Clinical Features: A 60-year-old man was diagnosed with Parkinson's disease at age 53 after a twitch developed in his left fifth finger. He later developed rigidity in his left leg, body tremor, slurring of speech, and memory loss among other findings.

Intervention and Outcome: This subject was managed with upper cervical chiropractic care for 9 months. Analysis of precision upper cervical radiographs determined upper cervical misalignment. Neurophysiology was monitored with paraspinal digital infrared imaging. This patient was placed on a specially designed knee-chest table for adjustment, which was delivered by hand to the first cervical vertebrae, according to radiographic findings. Evaluation of Parkinson's symptoms occurred by doctor's observation, the patient's subjective description of symptoms, and use of the Unified Parkinson's Disease Rating Scale. Reevaluations demonstrated a marked improvement in both subjective and objective findings.

Conclusion: Upper cervical chiropractic care aided by cervical radiographs and thermal imaging had a successful outcome for a patient with Parkinson's disease. Further investigation into upper cervical injury as a contributing factor to Parkinson's disease should be considered. (J Manipulative Physiol Ther 2000;23: 573-7)

Key Indexing Terms: Cervical Spine; Chiropractic; Parkinson's Disease; Trauma; Thermography

INTRODUCTION

A total of 1.5 million Americans have Parkinson's disease (PD), more than are afflicted with multiple sclerosis and muscular dystrophy combined.[1] Although PD is generally considered a disease that targets older adults, 15% of patients are diagnosed before age 50.[1]

PD, a progressive disorder of the central nervous system, results from destruction of the substantia nigra. The substantia nigra signals the basal ganglia (caudate nucleus and putamen) to secrete dopamine. Because dopamine is an inhibitory neurotransmitter, it is thought that the lack of dopamine allows the basal ganglia to send continuous excitatory signals to the corticospinal motor control system. Therefore overexcitation of the motor cortex (caused by lack of inhibition) creates typical Parkinson's symptoms such as rigidity (muscle tone increase) and tremors.[1] Current evidence suggests that PD symptoms appear after there has been an 80% loss of the dopamine-producing cells in the substantia nigra and a similar loss of dopamine synapses with the basal ganglia.[1]

Diagnosis of PD occurs through patient history and neurologic examination and is best determined by a physician specializing in movement disorders. No definitive laboratory test exists to diagnose or predict PD.

PD symptoms often begin with an episodic tremor of the hand on one side of the body. Over time, resting tremors can be accompanied by slowness, stiffness, and lack of arm swing on the affected side. As symptoms progress, impairment may extend to the other side of the body. Because of fine motor deficits, finger and hand movements requiring skilled coordination, such as brushing teeth, buttoning clothes, and handwriting, may become slow and difficult. Patients may notice a foot drag on the affected side, a slowed gait, shorter steps, or freezing (inability to start) when initiating movement. Voices may lose volume and facial expressions may become masked.

The standard medical treatment for PD has been the administration of a combination of levodopa (a short-acting drug that enters the brain and is converted into dopamine) and carbidopa (enhances levodopa's action in the brain). Several neurosurgical techniques also exist, including thalamotomy (destruction of ventral thalamus to control tremor), pallidotomy (destruction of posterior ventral globus pallidus to control hyperkinetic symptoms), and deep brain stimulation (electrode implantation for patient-controlled stimulation of thalamus to control tremor).[1] Although the medications and surgeries may temporarily control symptoms, they neither stop nor reverse the progressive degeneration of the substantia nigra.

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Palmer[2] reported treatment of patients with PD with upper cervical chiropractic care as early as 1934. In his writings, he referred to patients with "shaking palsy" and listed improvement or correction of symptoms such as "tremor, shaking, muscle cramps, muscle contracture, joint stiffness, fatigue, incoordination, trouble walking, numbness, pain, inability to walk, and muscle weakness."[2] His treatment included paraspinal thermal scanning with a neurocalometer, a cervical radiographic series to analyze the upper cervical spine, and a specific upper cervical adjustment performed by hand on a knee-chest table.

No other reference for the chiropractic management of PD was found. To my knowledge, this is the first report on this topic in recent decades.

CASE REPORT

A 60-year-old man first experienced PD symptoms at age 53 when his left fifth finger began to twitch. His neurologist diagnosed the patient with PD and prescribed medications, including carbidopa/levodopa, selegiline hydrochloride, and pramipexole dihydrochloride. Every 6 months, his neurologist monitored his condition and increased medication dosages as his condition worsened. Three years after the diagnosis, the patient's left leg became rigid, causing difficulty with walking. Most of the progression of PD symptoms occurred in the last 18 months before upper cervical chiropractic treatment.

Parkinson's symptoms were evaluated by doctor's observation, patient's subjective description of symptoms, and use of the Unified Parkinson's Disease Rating Scale (UPDRS).[3] The UPDRS was chosen over the Hoehn/Yahr and Schwab/England scales because the latter two provide only 5 and 10 staging categories, respectively. Conversely, the UPDRS classified 44 individual Parkinson's symptoms on a scale of 0 to 4, allowing for more detailed symptom comparisons during treatment. The 44 symptoms were rated during "on" and "off" stages of medication use, as directed by the scale's authors. An "on" stage occurred when medications temporarily decreased or masked Parkinson's symptoms; during an "off" stage, medications lost their effectiveness, so the true symptoms of the patient were exhibited. This patient took multiple medication dosages per day in an attempt to reduce the frequency and severity of "off" periods.

The UPDRS entrance symptoms of the patient, such as tremor, rigidity, and depression are illustrated in Table 1. Each symptom was rated from 0 to 4 according to disability level. A score of 0 indicated absence of the symptom, whereas 4 represented complete disability. The authors of the scale developed specific rating criteria for each symptom. For example, when evaluating falling using the rating scale, "0" indicated none, "1" denoted rare falls, "2" signified less than 1 fall per day, "3" represented 1 fall per day, and "4" indicated more than 1 fall per day. Thus if a patient were completely disabled in all symptom categories, he or she would score a 4 in each of the 44 categories, producing a total of 176 (44×4).

This patient's initial UPDRS evaluation was 32 during on stages and 74 during off stages, which is depicted as 32/74 (Table 1). His most severe symptoms included memory loss, depression, loss of motivation, slurred speech, illegible handwriting, tremor and rigidity in his left extremities, and difficulty arising from a chair. In addition to the symptoms rated by the UPDRS, he also experienced extreme fatigue, insomnia, and pain throughout his spine. The absence of these symptoms from the UPDRS reduced its effectiveness as a comparative tool; however, the UPDRS was the most comprehensive scale found.

Paraspinal digital infrared imaging, which measures cutaneous infrared heat emission, was chosen as the diagnostic test for neurophysiology. Thermography has been shown to be valid as a neurophysiologic diagnostic imaging procedure, with more than 6000 peer-reviewed and indexed articles over the past 20-year period. In blind studies comparing thermographic results with those of computed tomography scans, magnetic resonance imaging, electromyography, myelography, and surgery, thermography was shown to have a high degree of sensitivity (99.2%), specificity (as high as 98%), predictive value, and reliability.[4-6] Thermal imaging has been effective as a diagnostic tool for breast cancer, repetitive strain injuries, headaches, spinal problems, temporomandibular joint conditions, pain syndromes, arthritis, and vascular disorders, to name a few.[7-16] This is the first case reporting use of thermal imaging for a patient with PD.

At the patient's first chiropractic office visit, a paraspinal thermal analysis was performed from C7 to the occiput, according to thermographic protocol.[17-19] Compared with established normal values for the cervical spine, the patient's paraspinal scan contained thermal asymmetries as high as 1.13°C. According to cervical thermographic guidelines, thermal asymmetries ≥0.5°C indicate abnormal autonomic regulation or neuropathophysiology.[20-23]

Because upper cervical misalignments were suspected, a precision upper cervical radiographic series, including lateral, anterior-posterior, anterior-posterior open mouth, and base posterior views, was performed.[24] These 4 views enabled examination of the upper cervical spine in 3 dimensions: sagittal, coronal, and transverse. To maintain postural integrity, the patient was placed in a positioning chair with head clamps. Analysis of the 4 views was directed to the osseous structures (foramen magnum, occipital condyles, atlas, and axis) that are intimately associated with the neural axis. Laterality and rotation of atlas and axis were measured according to each vertebrae's deviation from the neural axis.[24] Right laterality of the atlas was found.

Cervical range of motion testing was painful on left lateral bending and left rotation. Left lateral flexion compression was positive. In this patient's lumbar spine, flexion, right rotation, and left lateral flexion produced pain.

Because the 2 criteria determining subluxation (thermal asymmetry and vertebral misalignment) were met, a treatment plan was discussed with the patient. After he gave consent, treatment began with an adjustment to correct the right laterality of atlas. To administer the adjustment, he was placed on a

knee-chest table with his head turned to the right. The knee-chest posture was chosen because of accessibility of anatomy. In addition, this posture retained spinal curvatures, thus preventing compression of the spine. With the right posterior arch of atlas as the contact point, an adjusting force was introduced by hand.[25] The adjustment's force (force = mass × acceleration) was generated with body drop (mass) and a toggle thrust (acceleration).

Then the patient was placed in a recuperation suite after the adjustment for 15 minutes, according to thermographic protocol.[17-19] The adjustment's success was determined by reviewing of the thermal scan after adjustment. The first scan after adjustment revealed a thermal difference of only 0.1°C, which was considered normal according to established cervical thermographic guidelines (compared with the differential before adjustment of 1.13°C).[20-23] Therefore resolution of the patient's initial thermal asymmetry was achieved.

All subsequent treatment visits began with a thermal scan. An adjustment was administered only when thermal asymmetry was present. If an adjustment was given, a second scan was performed after a 15-minute recuperation period to determine whether restoration of normal thermal symmetry had occurred.

His treatment visits occurred 3 times per week for the first 2 weeks of care. After the first adjustment, subsequent adjustments were administered on visits 2, 4, and 6. By the end of the second week of care, he reported greater range of motion in his neck, improved sleep, better energy, and decreased stiffness in his body overall.

During weeks 3 and 4 of care, visits were reduced to 2 times per week, and only 1 adjustment was administered during that time. A reevaluation occurred at the end of week 4. Cervical and lumbar ranges of motion no longer produced pain. Cervical compression test results were negative. The UPDRS reevaluation revealed a reduction in symptoms to 20/56 during on/off stages (Table 1). The patient reported that his most noticeable improvements included improved sleep and increased energy. He was more alert and was no longer tired or depressed. He had improved range of motion in his neck, better balance, improved hand and leg agility, and less rigidity overall. His left leg no longer dragged, and his walking improved. He routinely reported "feeling great." The symptoms of mental clarity, handwriting, turning in bed, and arising from a chair also improved.

During the next 8 weeks of care, the patient was seen 1 time each week and received an adjustment on 2 of 8 visits. At week 12, a final UPDRS reevaluation occurred, which revealed another reduction in Parkinson's symptoms to 13/47 during on/off stages (Table 1). During the third month of care, he reported that his greatest improvement was the return of his balance, which enabled him to resume riding a bike. He also noted that his wife, daughter, son, friends, and neighbors all noticed a marked improvement in his physical and mental health.

According to a comparison between beginning and final UPDRS evaluations, this patient showed an overall improvement of 43% after the third month of care (Table 2). To calculate the percentage, the total of the final evaluation (13 + 47 = 60) was subtracted from the initial evaluation (32 + 74 = 106), producing a difference of 46. This reduction of 46 points was divided by the original total of 106, yielding a 43% improvement. Although the UPDRS was helpful in evaluating specific Parkinson's symptoms, it did not take into consideration other associated symptoms, such as spinal pain, insomnia, and fatigue. Thus the scale underestimated both the patient's severity of symptoms at the beginning of treatment and his improvement after treatment. As a

Table 1. *United Parkinson's Disease Rating Scale (UPDRS)*

Symptom category	Week 1 (on/off)	Week 4 (on/off)	Week 12 (on/off)
Memory loss	2/4	2/3	1/2
Hallucinations	0/0	0/0	0/0
Depression	1/3	1/2	1/2
Loss of motivation	1/3	1/3	1/0
Slurred speech	1/4	1/2	1/2
Increased salivation	0/0	0/0	0/0
Swallowing/choking	0/0	0/0	0/0
Illegible handwriting	1/4	1/3	1/3
Cutting food	0/0	0/0	0/0
Dressing: buttons	1/2	1/2	0/1
Hygiene: brushing teeth	0/1	0/1	0/1
Turning in bed	2/2	1/2	0/1
Falling	1/2	1/2	0/1
Freezing	1/2	1/2	1/2
Walking	1/2	0/1	0/1
Tremor: entire body	1/2	1/2	1/2
Sensory: numbness, tingling	1/2	0/1	0/1
Monotone speech	2/4	0/2	0/1
Masked facial expression	1/2	0/2	0/2
Tremor: face, lips, chin	0/0	0/0	0/0
Tremor: right hand	0/0	0/0	0/0
Tremor: left hand	2/4	2/4	2/4
Tremor: right foot	0/0	0/0	0/0
Tremor: left foot	2/4	2/4	2/4
Action tremor: right	0/2	0/2	0/2
Action tremor: left	0/2	0/2	0/2
Rigidity: neck	0/0	0/0	0/0
Rigidity: right arm	0/0	0/0	0/0
Rigidity: left arm	1/3	1/2	1/2
Rigidity: right leg	0/0	0/0	0/0
Rigidity: left leg	1/3	1/2	1/2
Finger taps: right	0/0	0/0	0/0
Finger taps: left	1/2	0/0	0/0
Hand grips: right	0/0	0/0	0/0
Hand grips: left	1/2	0/1	0/1
Hand pronate/supinate: right	0/0	0/0	0/0
Hand pronate/supinate: left	1/2	0/1	0/1
Leg agility: right	0/0	0/0	0/0
Leg agility: left	1/2	0/1	0/1
Arise from a chair	2/3	0/1	0/1
Stooped posture	1/2	1/2	0/1
Postural stability	0/0	0/0	0/0
Gait	1/2	1/2	0/1
Body bradykinesia	1/2	1/2	1/2
Totals	32/74	20/56	13/47

Table 2. *Percent improvement*

Initial UPDRS (on/off)	Final UPDRS (on/off)	Present improved after 3 months*
32/74	13/47	43

*UPDRS did not evaluate spinal pain, fatigue, or insomnia; therefore percent improvement is understated.

UPDRS, United Parkinson's Disease Rating Scale.

result, his overall percent improvement after 3 months of treatment was underestimated.

Because of his spine's stability after 3 months of care, his treatment plan was reduced to 1 visit per month for the next 6 months. Adjustments were necessary on 2 visits. Over the 6-month period, he reported maintenance of his previous improvements and no deterioration in his condition. He also reported a continued gradual increase in energy level and strength in his body, as well as a continued reduction in muscle and joint stiffness. Consequently, between months 8 and 9, he enlisted a personal trainer's help and began an exercise program that included cardiovascular exercise and weight training 3 times per week. At the time of writing, he had undergone 9 months of upper cervical chiropractic care and intended to continue his maintenance treatment plan of 1 visit per month.

DISCUSSION

An important aspect of this patient's medical history was his recollection of head and/or neck traumas before the onset of PD. He recalled 6 specific incidences of trauma preceding the onset of symptoms, including 2 concussions while playing football, twice hitting his head against a windshield (during a helicopter crash and an auto accident), a sledding accident in which his legs were paralyzed for 24 hours, and a riding accident in which he was thrown from a horse. The body of medical literature detailing a possible trauma-induced cause for PD, or at least a contribution, is substantial.[26-31] In fact, medical research has established a connection between spinal trauma and numerous neurologic conditions besides PD, including but not limited to multiple sclerosis, epilepsy, migraine headaches, vertigo, amyotrophic lateral sclerosis, and attention deficit/ hyperactivity disorder.[22-28] Although medical research shows that trauma may lead to PD and the other neurologic conditions mentioned above, no mechanism has been defined. I hypothesize that the missing link may be the injury to the upper cervical spine.

Although various theories have been proposed to explain the effects of chiropractic adjustments, a combination of 2 theories seems most likely to explain the profound changes seen in this patient with PD after he received upper cervical chiropractic care. The first mechanism, central nervous system facilitation, can occur from an increase in afferent signals to the spinal cord and/or brain coming from articular mechanoreceptors after a spinal injury.[39-43] The upper cervical spine is uniquely suited to this condition because it possesses inherently poor biomechanic stability along with the greatest concentration of spinal mechanoreceptors.

Hyperafferent activation (through central nervous system facilitation) of the sympathetic vasomotor center in the brainstem and/or the superior cervical ganglion may lead to the second mechanism, cerebral penumbra, or brain hibernation.[44-50] According to this theory, a neuron can exist in a state of hibernation when a certain threshold of ischemia is reached. This ischemia level (not severe enough to cause cell death) allows the cell to remain alive, but the cell ceases to perform its designated purpose. The brain cell may remain in a hibernation state indefinitely, with the potential to resume function if normal blood flow is restored. If the degree of ischemia increases, the number of functioning cerebral cells decreases and the disability worsens.

It is likely that this patient sustained an injury to his upper cervical spine (visualized on cervical radiographs) during one or more of the traumas he experienced. It is also likely that because of the injury, through the mechanisms described previously, sympathetic malfunction occurred (measured by paraspinal digital infrared imaging), possibly causing a decrease in cerebral blood flow. If blood supply to this patient's substantial nigra was compromised, it is possible that a certain percentage of those cells were existing in a state of hibernation rather than cell death. Therefore the combination of theories suggests that when blood supply was restored to the hibernating substantial nigra cells (from upper cervical chiropractic care), the cells resumed their dopaminergic (dopamine-secreting nerve fibers) function. However, few conclusions can be drawn from a single case. Indeed, this patient was treated with upper cervical chiropractic along with 9 other patients with PD during a 3-month period. Therefore further research is recommended to study the links among trauma, the upper cervical spine, and neurologic disease.

CONCLUSION

This case report described a successful outcome for a patient with PD who was treated with upper cervical chiropractic care. To my knowledge, this is the first case reported on this topic since Palmer's research 70 years ago.[2] No firm conclusion can be obtained from the results of one case, although these results do suggest that upper cervical chiropractic care may provide benefit for patients with PD when an upper cervical injury is found. Further investigation into upper cervical injury and resulting neuropathophysiology as a possible cause or contributing factor to PD should be considered.

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