Case Report

| Med Cases. 2022;13(3):140-144

Mitigating Gait Decline in a Woman With Parkinson's Disease: A Case Report

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Abstract

Levodopa therapy is the standard pharmacological treatment for Parkinson's disease (PD). However, after an initial period of significant benefit, the effects of levodopa begin to wear off. This results in a reduction in the effect duration and the development of motor complications. We describe the case of a 69-year-old woman presented with a 3-year history of lower back pain and progressive left leg weakness. One year prior to referral for neurological assessment, the patient first noted progressive leg weakness and insufficient strength to rise from a chair. The diagnosis of PD was made after excluding potential neurological disorders. The patient was initially started on oral levodopa, which improved her motor symptoms considerably during the first year. However, dose adjustment and combined pharmacological strategies failed to sufficiently control motor symptoms during the subsequent year. The patient experienced declines in gait ability, clumsiness in the left limbs, and difficulty in performing housework. The patient then sought chiropractic attention. Gait rehabilitation was the major goal in the treatment program for this patient, with the impression of motor complications of PD. The intervention consisted of spinal manipulation, intermittent motorized traction of the lumbar segments, and gait training programs. Following 3 months of the intervention, the patient demonstrated increased muscle strength and improved gait characteristics, as depicted by a gait cyclogram and vertical ground reaction force graphing. The current report illustrates that a multicomponent chiropractic approach may be used as an additional measure to mitigate gait decline in PD patients.

Keywords: Exercise rehabilitation; Gait cyclogram; Gait decline; Parkinson's disease; Spinal manipulation

Introduction

Dopamine replacement therapy using the precursor levodopa

Manuscript submitted November 21, 2021, accepted January 5, 2022 Published online March 5, 2022

doi: https://doi.org/10.14740/jmc3856

(L-DOPA) is the standard treatment for Parkinson's disease (PD). Over time, following a continued degeneration of dopaminergic neurons [1], the brain begins to rely on the most recent dose of medication. With both weaning and lack of response, dopaminergic therapy demonstrates reduced effect when it has been used for a certain duration [2]. Gait problems, such as freezing of gait, reduced balance, and postural control, become more evident. Continuous stimulation (using device-aided therapies or deep brain stimulation) may help in controlling motor fluctuations, dyskinesias, and cardinal motor symptoms [3]. However, each advanced therapy has both advantages and disadvantages, and not all patients are suitable for these therapies. Gait problems remain an issue, thereby, warranting additional therapeutic interventions.

The current study serves as an example of mitigating gait impairment and restoring postural control in patients with PD. Some musculoskeletal problems can be treated with different types of manual therapy to improve muscle strength, balance, and joint mobility. Spinal manipulation and exercise-based interventions are examples of alternative options to address the limitations of dopaminergic therapy. Gait variability, the stride-to-stride fluctuations in walking, is a quantifiable feature of gait that alters with disease, in addition to the effects of therapeutic interventions and rehabilitation [4]. Here, we review contemporary insights into gait improvement following rehabilitation interventions.

Case Report

Investigations

A 69-year-old female presented with a 3-year history of progressive left leg weakness and unsteadiness of gait. Three years prior to our diagnosis, the patient presented with weakness of the left leg, slowing during walking, insufficient strength to rise from a chair, and lower back pain during a visit to her family physician. The patient was subsequently diagnosed with lumbar spondylosis. With no clear improvement following medication and physiotherapy for motor symptoms for 12 months, the patient was then referred to a neurologist. She was diagnosed with PD according to the Movement Disorder Society clinical diagnostic criteria [5]. Oral levodopa treatment was prescribed, and the patient responded well during the first year.

Following 1 year of treatment, the patient began to feel that the effects of her medication were increasingly short-

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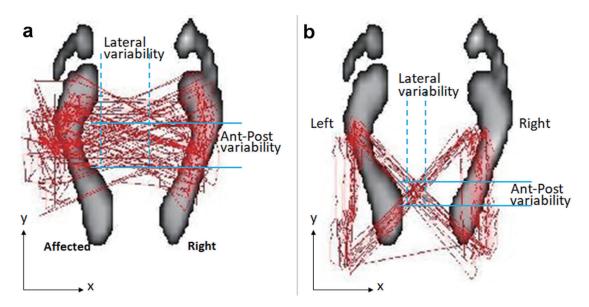


Figure 1. Walking cyclogram depicting notable improvement in gait characteristics after treatment. The traces (red butterfly plots) represent the trajectory of center of foot pressure on the instrumented treadmill. (a) Initially, the cyclogram showed a distorted butterfly wing feature, with a wider based gait, and great variability of stride and foot placement accuracy (blue intersected area). The disturbance was more pronounced on the left side. (b) After 3 months of intervention, the repeated cyclogram showed a more accurate foot stride, regular heel strike and symmetric and rhythmic center-of-pressure crossover between strides.

lived. Leg weakness, bradykinesia, and postural instability progressed over the next 12 months, despite repeated dose adjustments and combined pharmacological strategies. Aggravation of motor impairment progressed, which affected the left upper limb. The patient experienced difficulties in performing housework and became increasingly frustrated by her motor symptoms. As such, she sought chiropractic attention for her back pain and motor symptoms.

Diagnosis

At presentation, the patient exhibited slowing and walked cautiously with an irregular wide-based gait. Resting tremor was observed in her left hand and left leg. The patient's self-reported peak pain intensity of her back pain was 4 out of 10 on an 11-point numeric pain rating scale. Physical examination showed stiffness in the lower back, tenderness to palpation over the lumbosacral spine, reduced muscle strength of the left hip flexors and knee extensors, and hypertonicity of the left soleus and hamstring muscles. The patient's sensorium and cognition were intact. Motor strength measurements with a handheld dynamometer were graded at 4/5 of the left hand and 3/5 of the left leg. A walking trajectory (butterfly cyclogram) [6] depicted irregular stride length, great variations in stepping location, and a wider base of support (Fig. 1a).

Based on medical history and results of the test and measures, it was established that the patient had symptoms of PD motor complications. The primary medical International Classification of Diseases, Tenth Edition (ICD-10) codes were G20 (Parkinson's disease) and M47.816 (spondylosis without myelopathy or radiculopathy, lumbar region), and the primary physical therapy ICD-10 code was R26.9 (unspecified abnor-

malities of gait and mobility).

Treatment

The treatment plan was aimed at maintaining the patient's function whilst increasing muscle strength, balance, and gait ability, all of which would give potentially the patient the confidence to carry out tasks. Chiropractic intervention consisted of lumbar spine manipulation and intermittent motorized lumbar traction to release intersegmental restriction. Additionally, therapeutic ultrasound and massage to provide muscle flexibility, along with biceps curl balance and balance board training to retrieve balance were used. The frequency of treatment was twice weekly. Furthermore, home exercise recommendations included heel toe raises, seated marching, weight shifting, side-lying leg raise, and back bridging, in an attempt to improve walking ability.

Follow-up and outcomes

Following 3 months of the intervention the patient showed improvements in back pain and motor symptoms. This was shown by a decrease in pain scores (from 4 to 0 on an 11-point numeric rating scale) and an increase in dynamometer strength readings (an increase to 4+/5 in left hand and to 4/5 in left leg muscle strength). Additionally, an improvement in gait characteristics was shown by a more accurate foot stride, a regular heel strike and a symmetric and rhythmic center-of-pressure crossover between strides as depicted by a walking cyclogram (Fig. 1b). Furthermore, an improvement in foot pressure and a regular stance phase were recorded as vertical ground reaction

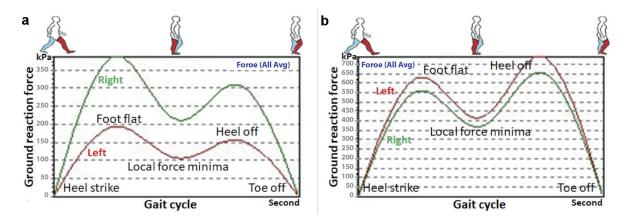


Figure 2. Graph comparing the gait pattern on vertical ground reaction force (GRF). (a) At initial presentation, the patient demonstrated a marked decrease in applied force of the left foot in the stance phase of the gait cycle, as compared to the right foot. (b) The follow-up vertical GRF graph recorded symmetric foot pressure and regular stance phase. It could be noted that the GRF peaks were markedly increased after treatment. The data represent the average vertical force (in kPa) of multiple stride cycles recorded by pressure foot sensors as the patient walked for 2 min on the instrumented treadmill.

force (GRF) graphing [7] (Fig. 2). The patient has been followed up for 12 months, and she continued her normal daily activities. No treatment-related adverse events were reported.

Discussion

The mechanism by which a patient's response to levodopa changes during long-term therapy is not fully understood. When the motor signs of PD emerge, approximately 20-40% of dopaminergic neurons may be remaining [8]. Presumably, the ingested levodopa is taken up by the remaining dopaminergic neurons and is converts to dopamine, which is stored and slowly released into the synaptic cleft. However, as the degeneration of dopamine neurons progresses, this conversion, storage, and release mechanism is compromised [8]. Plasma levels of levodopa will show extreme variations, and the response to levodopa become unpredictable, resulting in the appearance of motor fluctuations [1]. When gait decline cannot be effectively solved by pharmacological means, additional interventions to mitigate gait impairment and its progression are warranted [2]. However, exercise rehabilitation has become a promising adjunct treatment for PD [9].

Patients with PD display profound reductions in isokinetic muscle strength, which causes functional difficulties. The deficit in central activation [10] and the rate of force generation of muscles [11, 12] during repeated strengthening are responsible for muscle weakness. Increasing evidence suggests that exercise and motor training improves force-generating capacity, neuromuscular function, and functional performance [11, 13]. Spinal manipulation therapy, broadly speaking, is primarily used to treat musculoskeletal issues, with emphasis on a range of strategies to mobilize restricted structures and relieve neural compromise, and maximize the functions of contracted joints and affected muscles [4]. The positive changes observed after spinal manipulation and exercise-based training may be unrelated to treatment specificity but a systemic effect of functional changes in a biomechanical

interdependence [14]. It is speculated that pain relief following manipulative remedies may improve disc diffusion, neural proprioception of muscles, motor functions, and postural balance [4, 14].

Several factors may contribute to the optimization of gait ability in patients with PD. Gait impairment is the outcome of a complex interaction of disease progression, aging changes, compensatory mechanisms, and eventual secondary deconditioning due to restricted mobility. Speculatively, exercise-based interventions aimed at increasing muscle strength and activity may, at least partially, target age-related changes (sarcopenia, physical inactivity, osteoarthritis, etc.), which may positively impact PD gait [2]. Although the underlying mechanisms are multifactorial, sometimes all it takes is pain relief and gait improvement to increase a patient's motivation to continue, which may have led to even better outcomes. With regard to this case, by facilitating muscle strength and spinal excitability, chiropractic intervention itself might have substantially contributed to neuromuscular and mobility improvement in parkinsonian gait [4].

Researchers [15, 16] have observed that peripheral blood levels of brain-derived neurotrophic factor (BDNF) increased significantly after a single bout of aerobic or strength exercise, as well as after a training session. As a member of the neurotrophin family of growth factors, BDNF has a significant effect in promoting neurogenesis, synaptic plasticity, neuronal survival, and reducing inflammation. BDNF acts as a protective agent for dopaminergic neurons and is a promoter of neuroplasticity [17]. Neuroplasticity refers to the capacity of the nervous system to modify itself in response to experience and injury [18]. Therefore, BDNF facilitates exercise-based recovery [19].

Individuals with gait dysfunction demonstrate increased stride-to-stride fluctuations, which are known as gait variability. PD patients with axial disability typically adjust their movement pattern to move slowly and safely to avoid falls or injuries [4]. A more variable step width may compensate for balance difficulties [2]. Measurements of gait cadence, sym-

metry, stride variables, and foot placement characteristics can be useful markers of disease progression and response to therapy. Instrumented treadmill analysis systems can be implemented, which can measure stepping paradigms during ambulation. The overlaid graphical display of dynamic foot placement [6] is an intuitive way to quantify subtle changes in walking difficulty during therapeutic interventions (Fig. 1).

This report provides novel evidence that patients with PD can become significantly more functional in a relatively short amount of time after spinal manipulation with motor training, demonstrating a decreased pain score, increased muscle strength measurement, and improved gait characteristics depicted on gait cyclogram and vertical GRF graphing (Figs. 1, 2). A focus on movement strategies may therefore lead to the development of useful therapies. While it is often difficult to generalize results from a single case to a larger population, the actual duration of sustaining improved outcomes following a course of manipulative intervention remains to be determined.

Conclusions

This case report provides new evidence and highlights the value of chiropractic intervention in making neuromuscular and mobility improvement in parkinsonian gait, especially when the problem cannot be effectively solved by pharmacological means. It contributes significantly to the existing practice in the treatment for PD.

Acknowledgments

None to declare.

Financial Disclosure

None to declare.

Conflict of Interest

None to declare.

Informed Consent

Written informed consent was obtained from the patient for case publication.

Author Contributions

Each author has individually been involved in and has made substantial contributions to conception and design, acquisition of data, analysis, interpretation of data, drafting and editing the manuscript.

Data Availability

The authors declare that data supporting the findings of this study are available within the article.

Abbreviations

BDNF: brain-derived neurotrophic factor; GRF: ground reaction force; ICD: International Classification of Diseases; PD: Parkinson's disease

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