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Monitoring and Graphing Selected Motor and Non-Motor Symptom Fluctuations in a 72-Year-Old Parkinson's Patient: A Caregiver Perspective

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Abstract

Parkinson's Disease (PD) is characterized by a spectrum of symptoms, including movement disabilities and non-motor neuropsychiatric, sleep, sensory, and autonomic complications. Fluctuations of symptoms in PD create an additional burden for patients and, due to difficulties in their recognition and tracking, impediments to optimal management of the disease. This case report details the daily monitoring and graphing of motor and non-motor symptoms in a 72-year-old Parkinson's disease patient. Following a reduction in the patient's carbidopa/levodopa dosage, a caregiver used a custom observational scale to chart symptom fluctuations over three weeks. The study finds that the medication adjustment correlated with an attenuation of symptom fluctuations, suggesting this caregiver-led method may be a useful tool for tracking treatment effects in PD management.

Keywords

Parkinson's Disease, Motor and Non-Motor Symptoms, Diurnal Symptoms' Fluctuations, Carbidopa/Levodopa

1. Introduction

Patients with Parkinson's Disease (PD) present with a wide range of symptoms, clinically categorized as motor and non-motor symptoms. The four cardinal motor features of PD, manifesting as movement disturbances grouped under the acronym TRAP, comprise 1) tremor at rest (a pill-rolling movement); 2) rigidity of

muscles, felt by the examiner as increased muscle tone when palpated; 3) akinesia, or bradykinesia, characterized by slowness; and 4) postural instability. Secondary motor symptoms PD patients may experience include shuffling gait, festination and freezing of gait, dysarthria, dysphagia, and micrographia [1] [2].

Non-motor features of PD may present as seemingly unrelated signs and symptoms affecting multiple faculties, with a debilitating effect on patients' quality of life and involving various organs, which complicates diagnosis and masks their true prevalence [3]. Across the spectrum of non-motor symptoms, four domains have been distinguished: (1) sleep disturbances, such as REM sleep behavior disorder and excessive sleepiness; (2) neuropsychiatric disorders, including depression, anxiety, cognitive impairment, and visual hallucinations; (3) dysautonomias, including constipation, sexual dysfunction, and pain; and (4) olfactory dysfunction, such as anosmia. In addition, susceptible patients may experience side effects of dopaminergic treatment manifesting as hypersexuality or disruption of the reward system, such as those observed in pathological gambling [4].

The third feature of PD, fluctuations of symptoms, affects a large population of patients undergoing prolonged therapy with levodopa, the dopamine precursor used for the treatment of PD, and manifests as oscillations in the intensity of certain motor and non-motor symptoms. More specifically, the following four states have been distinguished: 1) "on", in which the adequate level of levodopa allows for good control of symptoms, particularly the absence of dyskinesia (e.g., chorea, dystonia, freezing); 2) "wearing-off", or end-of-dose deterioration (e.g., intensifying tremor, stiffness, muscle cramping, increasing anxiety); 3) "on-off" swings, characterized by a sudden deterioration of movement (e.g., akinesia, freezing of gait); and 4) "off", marking an insufficient levodopa level and return of motor symptoms (e.g., severe dyskinesia, temporary immobility) [5].

Since PD is a progressively degenerative condition, it is essential to monitor and control symptoms by assessing the patient's status regularly. In clinical practice, the most widely used assessment scale for PD symptoms is the Movement Disorder Society-Revised Unified Parkinson's Disease Rating Scale (MDS-UPDRS), a comprehensive 50-question assessment comprising four sections: non-motor experiences of daily living, motor experiences of daily living, motor examination, and motor complications [6]. In addition, the Non-Motor Symptoms Questionnaire (NMS-Q), a 30-question patient-completed assessment, allows physicians to screen for non-motor symptoms [7].

However, the items collected by measurement instruments during patients' infrequent clinical appointments are based on historical recall and self-reports; thus, they may not precisely reflect a patient's condition [8]. Given the impact of the disease on cognitive faculties, particularly during "off" states, and the fluctuating nature of PD symptoms, an "outside" perspective of a caregiver utilizing a longitudinal study design with simple visual modeling may present a valuable addition to PD clinical tools. It may also offer the families and the patients an approximation of the various physical and mental modes that they navigate every day.

While graphs tracing selected motor features with wearable sensory devices have been included in research [9], and graph results of self-reported mood and motor fluctuations have been reported [10], no research based on the live monitoring of motor and non-motor fluctuations during patients' daily activities with subsequent graphing has been published.

I report my experience as a caregiver for a PD patient whose neurologist lowered the carbidopa/levodopa dose to address his complaint of insomnia and the family's concern regarding his gambling habit. The subsequent attempt to monitor and document daily fluctuations of other motor and non-motor symptoms resulted in graphs reflecting a subtle attenuation of post-adjustment symptom fluctuations.

2. Case Report

The patient in this report was a 72-year-old retired engineer/businessman living alone and independently but with the daytime assistance of a caregiver. He had been diagnosed with Parkinson's disease a few years prior, for which he had been treated with carbidopa/levodopa. His past surgical history was significant for decompression of L4 L5 two years prior, complicated by Staph infection and coma. This resulted in limited mobility of the lumbar area and weakness in the lower extremities; he was using a walker to support his posture and walking. The patient was evaluated for progression of PD every six months by his neurologist. He was seen on a regular basis by his geriatrician for general health status and by a psychiatrist for mental evaluation.

He appeared pleasant and alert; his voice was soft, and his facial expression had a somewhat bland and masked look. He denied being depressed and was frequently reluctant to take anti-depression medication. Family members reported the patient had been unable to smell for years, even though he himself denied anosmia. He complained of mild constipation and experienced episodes of orthostatic hypotension.

The patient's antiparkinsonian pharmacotherapy initially consisted of 100/400 mg of carbidopa/levodopa four times per day. To address concerns regarding compulsive gambling and insomnia, the patient's neurologist adjusted the dose to 75/300 mg three times and 50/200 mg once per day, for a total levodopa dose decrease from 1600 mg to 1100 mg per day. His other medications included venlafaxine (Effexor), cephalexin (Keflex), and pyridoxine (**Table 1**).

The patient sustained notable fluctuations of symptoms during the daytime. The most pronounced observed positive deviations from the base were mania-like episodes with hallucinations about an imaginary parking lot filled with cars allegedly owned by the patient and urgent casino visits fulfilling his gambling compulsion. The most negative deviation from the base manifested as blunted consciousness, with Hebrew, the patient's first language, spoken and dystonia of extended feet bilaterally. On occasion, the patient would slip into a semi-conscious state, during which he would, on occasion, whisper in English, "I do not want to live". Rapid

eye movements could be observed during the most negative deviations of cognition and speech.

Table 1. Medications.

Medication	Dose (mg)	Dose (mg)	Dose (mg)	Dose (mg)
Sinemet CR				
Pre-adjustment	100/400	100/400	100/400	100/400
Post-adjustment	75/300	75/300	75/300	50/200
Cephalexin	500	500	500	500
Venlafaxine	150		75	
Pyridoxine	25			
Time	**8:00 AM**	**12:00 PM**	**4:00 PM**	**8:00 PM**

3. Methods

I monitored the patient's symptoms and charted their fluctuations after the carbidopa/levodopa dose reduction, between 9 a.m. and 6 p.m. during his daily activities for three consecutive weeks. I was assessing the severity of symptom fluctuations using a scale designed for the purpose of this monitoring: -3 being the most severe "off" state, 0 being base/normal, and 3 being the most positive deviation from the base/normal state.

I defined the "on" state of Parkinsonian fluctuations as "the base". In this state, the pharmacotherapy allowed for effective control of motor and non-motor symptoms, and the patient presented with merely mild rigidity of the arms and legs, along with a minimally detectable festination of gait. His speech was quiet yet clear. His thought processes were coherent, memory and attention were grossly intact, with the first and the second language, Hebrew and English respectively, used effortlessly.

As outlined above, although the already existing tools for assessing the scope and severity of Parkinson's disease, such as the Movement Disorder Society-Revised Unified Parkinson's Disease Rating Scale (MDS-UPDRS), provide a comprehensive estimation of both motor and non-motor symptoms, their "static", historical nature does not reflect the dynamics of symptoms' character and oscillations. This need to capture the patient's condition in *statu nascendi*, as it was happening, in the case of this Parkinson's patient, was particularly important, as mood and movement variations tended to occur in clusters and within certain time-frames requiring a scale reflecting these variations and times of their occurrence.

Four categories of non-motor symptoms emerged as having the most pronounced effect on the patient's daytime condition: energy, affect, cognition, and speech. Each category was further broken down into levels according to its deviation from the base, with descriptive labels assigned for each level (**Table 2**). A general psy-

chiatric progress note was used as a blueprint for estimating affect, cognition, and speech. Affect represented the patient's observed manifestation of emotion, cognition expressed his thought processes, and speech described his modes of utterance. Walking was also monitored as a motor reference, and its levels were mapped. The graphs presented here are Excel-generated one-week composites of the daily graphs.

Table 2. Symptom categories and their descriptive gradations.

Gradient	Gait	Speech	Cognition	Energy	Affect
3	arrested	stuttered	hallucinations	hypered	frenzy
2	shaky	cadenced	irritated	surge	restless
1	accelerated	pressured	agitated	elevated	anxious
0	normal walk	paced	alert	basic	quiet
−1	hesitant	blurred	clouded	tired	flat
−2	stiff	mumble	confused	exhausted	resigned
−3	freezed	first language	stupor	slump	frightened

4. Results

Based on the times at which particular symptoms reached their highest and lowest amplitudes, two subtly distinguishable fluctuation patterns could be recognized. In the first pattern, gait, speech, and cognition appeared to ebb and flow at similar times. The peaks of these indicators oscillated around 10 a.m., 2:30 p.m., and 5:30 p.m. (Figure 1), which correlated with the carbidopa/levodopa peak concentration of approximately 2 hours. The most pronounced ascension of this group of symptoms occurred at 2 p.m., two hours after the patient received the second 300 mg levodopa dose, with accelerated gait, pressured speech, and agitation. This progressed around 3 p.m. to an arrested gait, stuttered speech, and a thought process

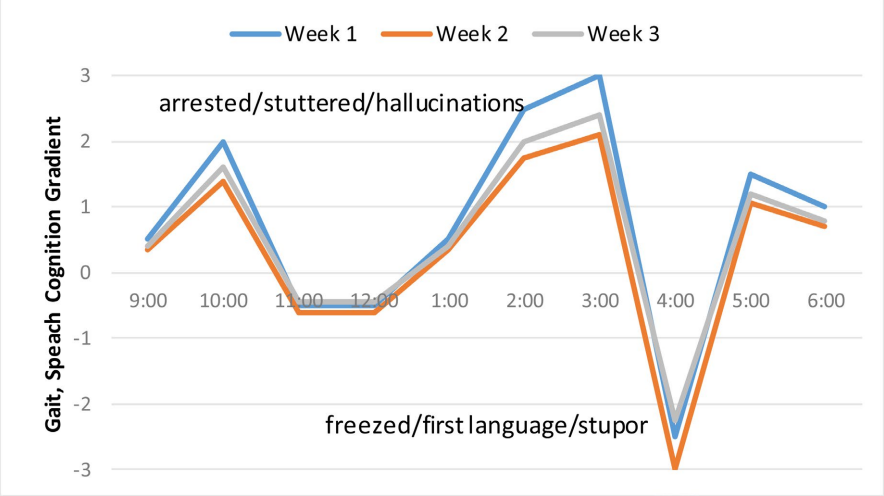

Figure 1. Fluctuations of gait, speech, and cognition.

characterized by irritation and an argumentative attitude.

Beginning around this time, the patient experienced a dramatic "swing" from peak to nadir in these categories: gait from arrested (peak) to frozen (nadir), speech from cadenced/stutter (peak) to first language (nadir), and cognition from irritated/contentious (peak) to stupor (nadir). Given the time frame (*i.e.*, one hour before the next dose of levodopa), this event had all the signs of the "wearing-off" effect, which persisted after the medication dose reduction. However, adjusting the dose of carbidopa/levodopa resulted not only in a decrease in predominantly positive deviations of symptoms but also reduced the frequency of use of first language and death ideations during much less severe semi-conscious episodes.

The second pattern was formed by fluctuations in energy and affect, which tended to move together. Two peaks could be observed: one at 12 p.m. and a more prominent one at 4 p.m. Both correlated with times of levodopa 300 mg intake. In addition, the most prominent positive symptom deviation coincided with a combined dose of 300 mg of levodopa and 75 mg of venlafaxine (Figure 2).

Figure 2. Fluctuations of energy and affect.

Around 3:30 p.m., the patient's energy and mood began deviating upward from the baseline, as manifested by a "hyper", manic-like state accompanied by rapidly increasing agitation and restlessness. This time interval, lasting from 30 to 60 minutes, correlated with a "swing" from peak to nadir in the three other categories: gait, cognition, and speech (Figure 3). This moment of clustering and confluence of symptoms between 3 p.m. and 4 p.m. was particularly uncomfortable for the patient, as the "stiffening" in gait, speech, and thought processing overlapped with rising anxiousness and restlessness.

After the carbidopa/levodopa reduction, I first observed a moderate decrease in the frequency with which the symptoms occurred. Most notably, the patient's hallucinations, manifesting as an urge to look for lost cars and a compulsion to visit casinos and gamble, became less persistent. He rarely spoke his first language during "off" states, and moments of mood depression, represented by dysphoria, also

became less conspicuous. Moreover, the graphs indicated that the adjustment resulted in the amelioration of the negative amplitudes of dysphoria, with less pronounced dyskinesias and relatively stable energy and mood.

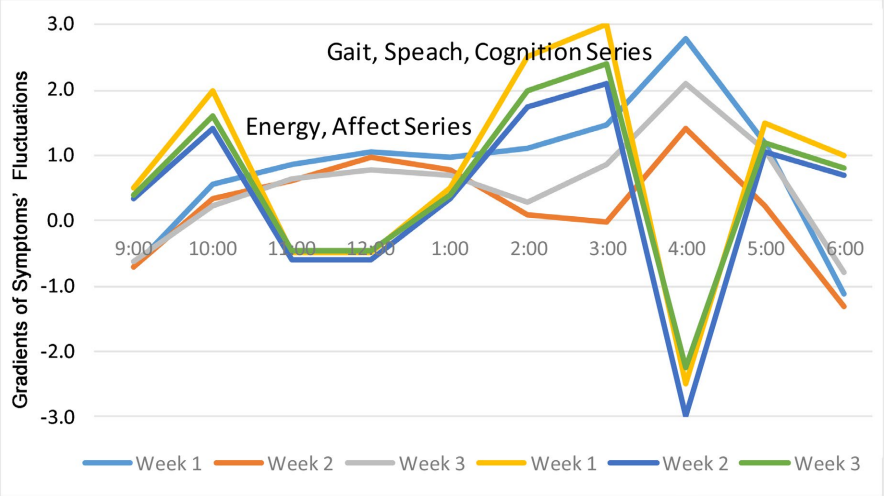

Figure 3. Confluence of Symptoms.

5. Discussion

This case demonstrates the results of monitoring and graphing symptom fluctuations in a Parkinson's patient following a reduction in his carbidopa/levodopa medication. Three sets of composite diurnal graphs prepared during three consecutive weeks post-dose reduction showed a moderate but noticeable correlation between the flattening of amplitudes in charted symptoms, which was particularly reflected in the patient's moderation of excessive affect, assuaging "off" dystonia and dysphoria, taming impulsive behavior, and modification of pharmacotherapy. These observations are consistent with research showing the benefits of adjustments in the levodopa regimen [11].

The patient presented with a wide range of symptoms reflecting a prolonged course of the disease and the likely impact of the levodopa treatment. Symptoms encompassed classic motor disabilities, including akinesia, rigidity, and postural instability, as well as festination and freezing of gait, and prominent non-motor features, such as fatigue, cognitive impairment, anxiety, depression, sleep disturbance, anosmia, and impulse control disorder, in the form of impulsive gambling that particularly affected the patient and his family. Crucially, some of these symptoms were complicated by their diurnal fluctuations, including the "on" and "off" phenomenon, which disrupted the patient's daily routine and amplified his sense of uncertainty.

The extensive list of PD symptoms reflects the disease's complex, multisystemic pathophysiology. Aside from additional degenerative processes, a number of particularly susceptible melanoneurons and other neural cells, damaged by the α-synuclein inclusions (Lewy bodies and Lewy neurites), lose the ability for neuro-

transmission in multiple neuronal networks. Following the six-stage topographic sequence, degeneration alters the flow of neurotransmitters along the visceromotor, somatomotor, limbic, and autonomic loops, which translates clinically to a disruption of various physiological and psychological functions, including olfactory impairment, deficits in responses to emotional stimuli, dysfunctions of visceromotor and endocrinal systems, and possible diminished cognitive faculties, with the development of dementia in some individuals [12].

The multisystemic complexity of PD pathophysiology is further complicated by symptom fluctuations, postulated to result from the long-term effects of levodopa pharmacotherapy. As the destruction of striatal dopaminergic nuclei responsible for motor deficits occurs, and the degenerative denervation of serotonergic, noradrenergic, and cholinergic nuclei responsible for non-motor pathology progresses, the neurons in the central nervous system lose their presynaptic storage capacity and postsynaptic response ability. Combined with the levodopa-impeded absorption in the periphery, the reduction of neural plasticity influences the plasma levodopa level and thus induces symptom fluctuations, further amplified by the levodopa nonphysiological pulsatile delivery [5].

Confirming this diverse pathophysiology of fluctuations, research evaluating their movements revealed a heterogeneous and complex pattern of non-motor symptom fluctuations that appeared to oscillate conjointly with motor symptoms, albeit without an obvious time-locked frequency. However, the mental/psychic symptoms exhibited a notable predilection for the "off" states linked to motor fluctuations; they tended to oscillate more frequently and severely in general, with fatigue, followed by the inability to concentrate, which appeared to impact PD patients the most [13]. Similarly, multiple patterns and the lack of a consistent mood/motor temporal relationship, possibly suggesting different underlying mechanisms, were found in a study tracing the diurnal movements of mood and motor fluctuations [10].

These findings mirror the wide array of motor, cognitive, and affective deficiencies manifested by the patient in this case, fluctuating with varying pace and intensity. Nevertheless, the fluctuations seemed to proceed in a quasi-synchronous manner, with peaks and troughs of energy and affect minimally preceding those of gait and cognition. Moreover, the conjoint movements of diurnal motor and cognitive symptoms may suggest a common basal ganglia pathophysiology, with affect and energy deriving their pathoetiology from a different mechanism.

Research has prominently featured the significance of the "on/off" and "off" states [13]. The graphs in the presented case confirm these results, with a particularly debilitating moment between 3 p.m. and 4 p.m., characterized by a surge in anxious affect, closely followed by an often unpredictably rapid progression from on-freezing to off-dystonia. Furthermore, the attenuation of the off-dystonia and dysphoria after the levodopa dose adjustment corroborates the notion that differentiation between non-motor fluctuations related to motor oscillations and non-motor symptoms, which are not influenced by on/off fluctuations, has therapeutic

implications. The former may be treated by adjustments of dopaminergic medication, whereas the latter may require specific symptomatic treatment [14].

Given this study's raw, exploratory character, it has various limitations, such as the lack of controls and the use of a non-standardized scale, which undoubtedly made its observations vulnerable to subjective bias in assessing the patient's states. Considering that the researcher was also the patient's caregiver, the potential for observer bias existed, and this dual role might have influenced the subjective symptom ratings and interpretation of improvement. In addition, the study did not include the role of factors other than medications, such as variations in daily routine or sleep over the three-week period, which could potentially have a confounding effect on variables. Nevertheless, the results collected not only validated a pharmacotherapeutic effect but were also helpful in predicting the course of daily fluctuations, which facilitated more efficient planning of the patient's activities. Therefore, a better understanding of the neurological mechanisms underlying the phenomenology of diurnal, minute fluctuations of the PD symptoms might not only enhance the clinical diagnostics, pharmacotherapy efficacy, and management of the condition, but could also provide potential input for the exploration of interactions between neurotransmitters involved in the PD pathophysiology. The practical application of caregiver-generated, dynamic charts for Parkinson's patients may also become a real possibility in clinical settings, as they could serve as a visual aid during appointments to supplement patient self-reports and guide treatment discussions.

Conflicts of Interest

The author declares no conflicts of interest regarding the publication of this paper.

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