

Management of a Patient with Depression and Parkinson's Disease

Ejona Shaska*

Department of Emergency Psychiatry, "Ali Mihali" Psychiatric Hospital, Vlora, Albania

Abstract

Background: Parkinson's disease impacts between 1 and 2 people per 1000 in everyday life. The incidence of this disease increases with age, and 1% of the population above 60 years is affected by Parkinson's disease.

Case report: We present the case of a 75-year-old female who was in the beginning diagnosed with Major Depressive Disorder (MDD) in 2019, based on DSM-5 criteria, clinical interview, and psychological assessment using the Beck Depression inventory. Two years later, she was diagnosed with Morbus Parkinson (Parkinson's disease). The therapy started with carbidopa-levodopa (Sinemet, Madopar) in combination with her psychiatric treatment plan, which was modified to include SNRIs and vitamin

supplementation. This case illustrates the frequent comorbidity between depression and Parkinson's disease, in which depressive symptoms frequently appear before motor symptoms.

Conclusion: This case presentation emphasized the importance of a multidisciplinary approach in patients with depression and Parkinson's disease.

Keywords: Parkinson's disease, depression, Morbus Parkinson, multidisciplinary approach

INTRODUCTION

Morbus Parkinson is a degenerative disease characterized by irreversible damage that affects the nervous system, mainly the neurons that produce the neurotransmitter dopamine in a specific area of the brain called the substantia nigra and in other pigmented nuclei of the brainstem. Parkinson's disease impacts between 1 and 2 people per 1000 in everyday life. The incidence of this disease increases with age, and 1% of the population above 60 years is affected by Parkinson's disease. Additionally, it has been found that 5% to 10% of cases have a hereditary tendency. More men than women develop this disease. As years pass, the prevalence and mortality of Parkinson's disease are increasing because people nowadays live longer, in combination with the lower mortality risk from other chronic diseases and cardiovascular diseases. So, they are more likely to live with the disease and die from its complications (1,2). PD causes motor symptoms including: slow movement (bradykinesia and hypokinesia), tremor, involuntary movement, rigidity, postural dysregulation, masked face, hypomimia. Non-motor symptoms include: cognitive impairment, mental health disorders, dementia, sleep and appetite disorders, pain, memory loss, decreased energy, difficulty concentrating and sensory disturbances, even sexual dysfunction. For evaluation, the Unified Parkinson's Disease Rating Scale measures behavior, mood, tremors, motor activation, patient's mental status, and therapy complications (3,4). The Movement

Disorder Society's criteria for Parkinson's disease recognize motor parkinsonism as the central characteristic, which is defined as bradykinesia with either resting motor or muscle rigidity (5). Other features include sleep dysfunction and REM sleep disorder, mood disorder, loss of smell, sialorrhea, and anosmia (6). But the best way to diagnose Parkinson's disease is a clear response to levodopa treatment (7). MRI imaging can help in differential diagnosis between Parkinson's disease and atypical Parkinsonism, especially now with the help of high-field MRI and new optimized MRI sequences. Also, it can exclude normal pressure hydrocephalus or subcortical stroke. Parkinson's disease intersects with other diseases, which include Parkinsonian variant of multiple system atrophy (MSA-P), progressive supranuclear palsy (PSP), and essential tremor (8).

CASE PRESENTATION

We present the case of a 75-year-old female who was in the beginning diagnosed with Major Depressive Disorder (MDD) in 2019, based on DSM-5 criteria, clinical interview, and psychological assessment using the Beck Depression inventory. The patient presented with motor symptoms such as bradykinesia, rigidity, trochlea dentata, hypomimia, mild tremor of the extremities, postural instability and was referred for consultation to a neurologist who performed a head CT and was diagnosed with Parkinson's disease. The Hoehn and Yahr scale was used to examine the severity of Parkinson's disease in the

patient, with a median stage of 2.5, indicating mild bilateral involvement with some postural instability. In the head CT, calcifications of millimetric size were found at the anatomical location of the basal ganglia bilaterally, most likely representing degenerative changes. Also, CT scan findings exclude acute or space-occupying lesions such as ischemic or hemorrhagic stroke, tumors, and abscesses. A mild, symmetric enlargement of the ventricular system and subarachnoid spaces indicates diffuse, age-related atrophy. She was given pharmaceutical treatment, which included benzodiazepines, antipsychotics, and SSRIs. Despite that, her condition worsened, and motor symptoms slowly appeared, like hypomimia, resting tremor, and muscle rigidity. Two years later, she was diagnosed with Morbus Parkinson (Parkinson's disease). Initially, she was treated with Madopar 250 mg (3x1/2 tab per day) for about 6 months and Lioresal 10 mg 2x1/2 tab per day, but the condition was unstable. Since she had rigidity, the neurologist accompanied her for a short period of time with Sinemet 25/100 2x1/2 tab per day, but discontinued it due to side effects (nausea, vomiting). The patient is currently taking Madopar 250 mg 3x 3/4 tab per day, Mirapexim 0.18 3x1 tab per day, Zoloft 50 mg 1 tab per day in combination with her psychiatric treatment plan, which was modified to include SNRIs (Duxet) and vitamin supplementation. Now she is in a stable condition. This case illustrates the frequent comorbidity between depression and Parkinson's disease, in which

depressive symptoms frequently appear before motor symptoms.

DISCUSSION

In the case presentation, we emphasized the importance of a multidisciplinary approach. With this approach, patients can boost their balance and their stability and have a more active life. Neurodegeneration affecting dopaminergic, serotonergic, and noradrenergic pathways likely contributes to early mood disturbances. Multidisciplinary treatment is essential to improve patient outcomes and quality of life in such cases. In terms of management and treatment of Parkinson's disease, the typical pharmaceutical treatment is levodopa with carbidopa, which reduces side effects and has better CNS bioavailability. For younger patients, usually a dopamine agonist (Ropinirole, Pramipexole) is used to prevent side effects, but it is less effective than levodopa, and generally, they need more aggressive therapy. In case the major symptom is tremor, it can be treated with Anticholinergics or Amantadine. Selegiline is useful for early disease and gives partial relief of symptoms. Up to 3 to 6 years, the treatment is effective. After that period, the disease is insensitive to medications. Patients with good responsiveness can exhibit time-resistance, on-off Phenomenon, or dyskinesia. To prevent this, we can alter the pharmacokinetics of levodopa, for example, the provision of delayed-release levodopa or continuous GI pump infusion. Also, there is a new approach that isn't fully

understood, and this is for some patients who are unresponsive. Deep brain stimulation can regulate the equilibrium of excitatory and inhibitory signals directed at the subthalamic nucleus or the globus pallidus (9-11). Additionally, Parkinson's disease has some serious complications. Autonomic dysfunction represents a significant non-motor feature of Parkinson's disease. In recent years, growing research interest has been directed toward its potential role in predicting and diagnosing PD at early stages, positioning it as a key area of investigation within the field. The spectrum of autonomic disturbances in PD encompasses gastrointestinal issues, cardiovascular irregularities, urinary problems, sexual dysfunction, abnormalities in thermoregulation, and impairments in pupil response and tear production (12,13). Another complication is Dementia. Dementia affects many patients with Parkinson's disease, with a prevalence of nearly 30%. The cognitive impairments involve low attention span, memory problems, executive dysfunction, and visuospatial difficulties. Behavioral symptoms can also be observed, such as apathy, visual hallucinations, and delusions. The most notable underlying pathology is the presence of Lewy bodies and a cholinergic deficit (14). As previously mentioned in the case, depression is a common complication of Parkinson's disease. Depression can appear before motor symptoms. Not only does it affect mood, but depression in PD can have other neuropsychiatric issues and late-stage

complications like dementia. Usually, it goes undiagnosed and inadequately managed, even though it affects the quality of life and cognitive function. The problem is not isolated in particular structural or functional brain abnormalities, but there is a widespread disruption in the communication between different brain regions. These disruptions are driven by pathological alterations in various neurotransmitter and receptor systems (15).

CONCLUSION

In conclusion, this case presentation emphasized the importance of a multidisciplinary approach in patients with depression and Parkinson's disease. With this approach, patients can boost their balance and their stability and have a more active life.

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