JNP

Diagnosis and Management of Cognitive Impairment in Parkinson's Disease

Alanna Dancis, MSN, CNP, and Valerie T. Cotter, DrNP, AGPCNP-BC

ABSTRACT

Originally considered a movement disorder that did not affect cognition, it is now understood that cognitive impairment is widespread among those with Parkinson's disease. Patients present with a variety of cognitive deficits, including executive dysfunction, visuospatial impairment, and memory loss. Neuropsychiatric symptoms are common, particularly psychosis, depression, and anxiety. Rivastigmine and donepezil have both been shown in large, blinded studies to slow the progression of dementia, although rivastigmine is the only medication approved by the United States Food and Drug Administration. Cognitive rehabilitation may be a safe alternative if medications are intolerable. Research is still needed at all levels to gain understanding of this disease process.

Keywords: cognitive impairment, dementia, geriatrics, movement disorder, Parkinson's disease

Parkinson's disease (PD) is a neurodegenerative disorder characterized by tremor, bradykinesia, rigidity, and postural instability. Although all PD patients have some degree of movement impairment, many patients have associated symptoms, including depression, psychosis, and cognitive impairment. Cognitive impairment is pervasive within the PD population. According to the Movement Disorder Society (MDS), an estimated 26.7% of PD patients have mild cognitive impairment,[1] whereas another 30% to 40% live with Parkinson's disease dementia (PDD).[2] One 20-year, longitudinal study suggested that up to 83% of PD patients will develop dementia in their lifetime.[3]

In this study we address much of the recent literature dedicated to the subject of cognitive impairment in PD. As evidenced by epidemiologic studies, knowledge of cognitive impairment in PD is relevant for all primary care providers, including nurse practitioners and physician assistants.

American Association of Nurse Practitioners (AANP) members may receive 0.78 continuing education contact hours and 0.28 pharmacology credit, approved by AANP, by reading this article and completing the online posttest and evaluation at cecenter.aanp.org/program?area=JNP.

PATHOPHYSIOLOGY

PD is caused by a depletion of the cells that produce dopamine within the substantia nigra. The dopamine produced in the substantia nigra is disseminated throughout the brain via the basal ganglia and plays a key role in both initiating and inhibiting movement.[4] The exact cause of PD is not known; some people who exhibit signs of parkinsonism have a genetic predisposition, whereas others have been exposed to certain toxic chemicals. However, in over 80% of those affected there is no known cause.[5]

Similarly, the exact mechanism of cognitive impairment in PD is not known. It is known that, over time, there is widespread brain atrophy and brain cell death in those with PDD. Cell death in PDD is largely attributed to the development of Lewy bodies. Lewy bodies are proteins that clump together for an unknown reason and then deposit in neurons causing cell damage and eventual cell death.[5] Lewy bodies also develop in Lewy body dementia (LBD), although in LBD the cognitive changes happen first. There is no consensus at this time as to whether PD and LBD are 2 distinct disease processes or variations of the same disorder.[6]

CLINICAL PRESENTATION

Case Study 1: Mrs. Black (MCI-PD)

Mrs. Black was diagnosed with Parkinson's disease 5 years earlier and began to exhibit signs of mild cognitive impairment in PD (MCI-PD) 3 years earlier. During the summer she and her husband had planned to attend a wedding, which was to take place in 3 weeks. Although she claimed to understand that the wedding was 3 weeks away, she would ask her husband daily, "Is the wedding today?" Later in the autumn her husband left the house for 30 minutes to buy dinner. Mrs. Black called her sister several times within those 30 minutes, thinking her husband had been gone for days. In addition, Mrs. Black fell frequently because she did not remember to ask her husband for assistance with ambulation, although she understood and verbalized the need for such help. Although she had some noticeable cognitive impairment, she still remembered the names of her family members, had lost no vocabulary, and read the newspaper each morning. As is common in the early stages of MCI-PD, it would not be clear from casual conversation with Mrs. Black that any cognitive changes had taken place.

Although PDD shares many of the same features with other types of dementia, the original clinical presentation can be quite different. If MCI progresses to Alzheimer's disease, memory impairment is almost always the presenting symptom.[7] In contrast, MCI-PD patients can present with memory, executive function, visuospatial, or language impairment.[8] Executive function impairment is a characteristic sign of cognitive impairment in PD. In executive function impairment, as is the case with Mrs. Black, patients will have difficulty planning and managing time. They will also have trouble shifting attention, reasoning, and overriding instinctual behavior.[9] A patient with visuospatial dysfunction may no longer have the ability to identify objects using vision, even if the eyes function properly. The presentation of MCI-PD is heterogeneous and deficits in any of the cognitive domains are relatively common. It is not unusual for an MCI-PD patient to present similarly to Mrs. Black, without memory or language impairments. For this reason it is important for the primary care clinician to comprehensively assess for all types of cognitive impairment.

Case Study 2: Mr. O'Henry (PDD)

Mr. O'Henry was diagnosed with PD 7 years earlier. He had advanced motor symptoms, including postural instability, rendering him unable to ambulate independently and he had difficulty swallowing. He was also diagnosed with PDD. He spent most of his days in bed, even though he maintained the physical ability to walk with assistance. He had hallucinations of insects in his bed, and maggots in his food. He believed his girlfriend was trying to sneak medications into his food to try to kill him. Therefore, it was difficult to persuade Mr. O'Henry to adhere to his carbidopa-levodopa regimen, worsening his movement symptoms.

Mr. O'Henry demonstrated some of the most devastating features of PDD: neuropsychiatric symptoms. Lee and colleagues, in their descriptive study, measured neuropsychiatric symptoms in 127 participants with PDD.[10] They found that 89% of their PDD study participants ($n = 113$) had at least 1 neuropsychiatric symptom; of these patients, 49.6% ($n = 63$) would hallucinate at least some of the time. This demonstrates how common psychiatric disturbances are in the PD population. Other common neuropsychiatric symptoms in PDD include anxiety, depression, and delusions.[2,10,11] However, in contrast to Alzheimer's disease, agitation, anger, and irritable mood are not common findings in PDD (Box 1).[2,7]

DIAGNOSIS—MDS CRITERIA

The first step in diagnosis of PDD or MCI-PD is to rule out other possible explanations for changes

Box 1. Clinical Presentation Pearls

- Assess patients for cognitive impairment who have Parkinson's disease. It is a common finding.
- Look for a wide array of clinical presentations (executive function impairment, visuospatial dysfunction, memory loss, language impairment). Memory loss may not be the presenting symptom.
- Assess for neuropsychiatric symptoms such as psychosis, depression, and anxiety.