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Parkinson Disease in a Patient with Multiple Co-morbidities: A Delayed Diagnosis?

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INTRODUCTION

Parkinson disease (PD) is a progressive neurodegenerative disorder with an estimated prevalence of 0.3% in the United States, while in those individuals 85 years and older, the prevalence increases to 4 - 5%. It is a condition that can be difficult to identify, especially in patients with multiple comorbidities, where signs and symptoms may overlap significantly.

CASE REPORT

A 64-year-old male with previous diagnoses of chronic inflammatory demyelinating polyneuropathy (CIDP), ulcerative colitis resulting in a remote partial small bowel resection, rheumatoid arthritis, and monoclonal gammopathy of unknown significance presented to the hospital with complaints of intractable abdominal pain, primarily post-prandial, and significant weight loss. His body mass index was 16 and vital signs were within normal limits. Review of symptoms revealed difficulty walking, frequent falls, tremor, and problems with memory. His physical exam revealed an abdomen mildly tender to deep palpation. He exhibited a stooped, lordotic posture and an unsteady, relatively wide-based gait. Routine labs indicated a stable anemia with hemoglobin of 11.9 g/dl. Esophagogastroduodenoscopy, colonoscopy, and gastric emptying study were unremarkable. An abdominal computer tomography (CT) scan showed possible bowel ischemia and vascular surgery was consulted. An abdominal CT angiogram (CTA) showed marked focal stenosis of the celiac artery origin, inferior mesenteric artery origin, and proximal to mid superior mesenteric artery. He was taken to the operating room for angioplasty and stent placement of affected vessels. His abdominal pain improved and his caloric intake increased sufficiently for discharge. At follow-up with neurology, the possibility of Parkinson disease (PD) was raised and he underwent

a DaTScan, which uses single photon emission computed tomography (SPECT) brain imaging to assist in the evaluation of
patients with suspected Parkinsonian syndromes. The DaTScan
showed no activity in the bilateral putamen and only faint activity in the bilateral caudate nuclei, with slightly more activity seen on the left. These findings were consistent with PD.
The patient recalled that symptoms initially began in 2006
with tremors in his right hand. It was thought to be an essential tremor, as several individuals in his family had the same.
Although he had several features not typical of PD, including
having a long history of tremors, severe CIDP, and the lack of
a typical resting tremor, he was started on carbidopa/levodopa
and his tremors and gait improved. While diagnosis and treatment of PD did not resolve every symptom, it improved the patient's function and safety, making this an important finding.

DISCUSSION

Parkinson disease is a progressive degeneration of the central nervous system, mainly affecting the motor system.² Characteristic neuropathologic features of the disease are dopaminergic neuron degeneration in the substantia nigra and the presence of eosinophilic intracytoplasmic inclusions (Lewy bodies) in the residual dopaminergic neurons. The etiology is likely multifactorial, with hereditary predisposition, environmental factors, and physiologic changes of aging all contributing. The disease encompasses a range of severity, from "parkinsonism", manifested by minimal, non-life-altering symptoms, to full-blown disease that greatly affects activities of daily living. PD generally presents with gait instability, changes in memory and cognition, slowed movement (bradykinesia), postural instability often resulting in falls, increased muscle rigidity or the development of masked facies. Lesser manifestations of these signs and symptoms can be subtle and difficult to recognize. Onset is often unilateral and may include other abnormal movements, such as postural or action tremors.

Patients may complain of insomnia, depression, anxiety, fatigue, constipation, dysautonomia, and anosmia.² Later in the disease, psychosis, involving visual hallucinations and delusions, and dementia occur in up to 25% of patients. A differential diagnosis must include Lewy body dementia, progressive supranuclear palsy (PSP), corticobasal degeneration (CBD), essential tremor, post-encephalitic conditions, and Alzheimer disease, as all can mimic PD.² Parkinson disease is the second most common neurodegenerative disorder, following Alzheimer disease, and is more common among Hispanics and non-Hispanic whites than Asians and African Americans.

The American Academy of Neurology (AAN) recommends initial treatment with levodopa or a dopamine agonist, depending on whether the need is to improve motor disability (levodopa is better) or decrease motor complications (dopamine agonists cause fewer motor complications), replacing endogenous dopamine in the form of levodopa, which is converted to dopamine in the brain.³ Levodopa is effective at controlling bradykinesia and rigidity.

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continued.

Levodopa is combined with carbidopa, which prevents peripheral conversion to dopamine by blocking dopa decarboxylase. Other effective agents which directly stimulate dopamine receptors include bromocriptine (Parlodel), pergolide (Permax), pramipexole (Mirapex), and ropinirole (Requip). Inhibitors of catechol-O-methyltransferase (COMT), including entacapone (Comtan) and tolcapone (Tasmar), decrease the breakdown of levodopa and extend its half-life, lessening the endof-dose wearing-off effect. Amantadine, an antiviral, provides benefits lasting for less than eight months, with its withdrawal resulting in a 10% to 20% rebound increase in dyskinesia.⁴ Anticholinergics can be used to treat the depression, dementia, and psychoses that develop in 20% to 40% of patients.⁵

The Unified Parkinson Disease Rating Scale (UPDRS; available at http://www.mdvu.org/pdf/updrs.pdf) is a standard assessment tool that provides a measure of disease progression and treatment response.⁶ The four-part scale measures mental effects, limitations in activities of daily living (ADLs), motor impairment, and treatment or disease complications.

Treatment of advanced or disabling symptoms can include deep brain stimulation of the subthalamic nucleus or globus pallidus.² Deep brain stimulation of the subthalamic nucleus effectively improves motor function and reduces motor fluctuations, dyskinesia, and antiparkinsonian medication use.⁷ Parkinsonism-directed physical therapy is often effective in reducing falls.

CONCLUSION

This case report illuminates that diagnoses often are delayed, especially when signs and symptoms overlap with other diseases. In a patient with multiple medical comorbidities, it can be challenging to diagnose this insidious disease.

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