


Two Cases of Parkinson Disease and Concurrent Myasthenia Gravis, Generalized and Ocular

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Leslie A. Neuman, MD¹ and Farooq Z. Cheema, MBBS²

Since 1987, to our knowledge, 10 cases of concurrent Parkinson disease (PD) and myasthenia gravis (MG) have been reported. We have cared for 2 patients with concurrent PD and MG and would like to share our experience with your readers and add these cases to the existing literature.

Case 1

In 2000, a 70-year-old woman presented with a 2-year history of right-hand resting tremor, stiffness, and slowness. Magnetic resonance imaging of the brain was negative and a presumptive diagnosis of PD was made. She responded well to carbidopa–levodopa. In 2005, she presented with the subacute onset of weakness of the limbs and neck flexor muscles. Ocular and bulbar muscles were spared. Routine laboratory studies, including thyroid function, were normal. Two weeks later, she developed increasing dyspnea and was hospitalized and intubated. Computerized tomography of the chest demonstrated bilateral pulmonary infiltrates. Thymic tissue or a mass lesion were not seen. Initially, it was felt that her respiratory failure was secondary to aspiration pneumonia as a complication of her PD. However, 10 days later, a paraneoplastic panel returned, revealing an acetylcholine receptor-binding antibody of 13.2 nmol/L (normal <0.02), striational antibody of 1:7680 (normal <1:60), and acetylcholine receptor-modulating antibody of 94% (normal 0%-20%). The other 11 antibodies in the panel were negative, including those against voltage-gated calcium channels. After having side effects to pyridostigmine and azathioprine, she was started on intravenous immunoglobulin (IVIG). She has been maintained on a program of 2 g/kg IVIG every 6 weeks. After 8 years, her muscle strength has been good with no recurrence of respiratory failure. Her PD has progressed but she remains ambulatory with the assistance of a cane. Her last acetylcholine receptor antibody titer was 0.49 nmol/L.

Case 2

In 2008, a 72-year-old man was seen by a neuroophthalmologist with ptosis and ophthalmoplegia. Laboratory studies

and imaging of the head and chest were normal except for an elevated acetylcholine receptor antibody titer. He has been treated with low-dosage pyridostigmine for ocular MG and has had no progression of clinical weakness. One year ago, he developed a resting tremor of the right arm with stiffness and slowness of the right arm and leg. Examination was consistent with early parkinsonism. He had mild ptosis and ophthalmoplegia but no bulbar or extremity weakness. In view of his mild PD motor findings, symptomatic therapy, such as carbidopa–levodopa, was not started. He was clinically stable at 6-month follow-up.

These are the first reported cases of severe generalized MG and ocular MG associated with concurrent PD. Previously reported cases of concurrent PD and MG had mild generalized weakness or isolated “head drop”¹⁻⁴. The presentation of our cases with ocular MG and myasthenic crisis differs and should alert clinicians to the range of MG presentations in patients with PD. Parkinson disease and MG share many motor and nonmotor clinical features including fatigue, weakness, dysarthria, dysphagia, and diplopia, which may mask or delay the diagnosis of MG. Whether or not there is more than a coincidental relationship between PD and MG is yet to be determined. Considering the relatively small size of our referral base, we suspect there are likely other concurrent cases of PD and MG in other Parkinson clinics.

We are reporting the severe generalized MG case to especially emphasize that one needs to keep an open mind in assessing respiratory failure in the patient with PD. It has been estimated that 9% to 45% of patients with PD die of pneumonia.⁵ One must be vigilant to not overlook another

¹ Department of Neurology, Hauenstein Parkinson Center, Mercy Health St Mary's, Grand Rapids, MI, USA

² Department of Pulmonary Medicine and Critical Care, Henry Ford Hospital, Detroit, MI, USA

Corresponding Author:

Leslie A. Neuman, Hauenstein Parkinson Center, Mercy Health St Mary's, 200 Jefferson SE, Grand Rapids, MI 49503, USA.
Email: neumla@mercyhealth.com

potentially treatable neuromuscular condition contributing to clinical deterioration in patients with PD. Motor power is not affected in PD and muscle weakness obligates a search for another neurologic explanation.

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