Case Report

An interesting cause of dysphagia in a patient with parkinson's disease

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Parkinson's disease is a neurodegenerative disorder and dysphagia is a frequent symptom of Parkinson's disease. We report a case of 61 year old male who presented with history of dysphagia, dysarthria, and diplopia of 6 weeks duration. He was a known case of Parkinson's disease for the past one year and was on levodopa. On examination he had bilateral ptosis and jaw weakness. Neostigmine test and acetylcholine receptor antibodies were positive. Repetitive nerve stimulation test was positive for decremental response. Hence diagnosis of myasthenia gravis was made. Dysphagia is a common manifestation of advanced parkinson's disease. But in patients in whom dysphagia does not improve with levodopa, one should keep possibilities of other etiologies for dysphagia in their mind and work up should be done to rule out other etiologies. We report this case to highlight a rare occurrence of Parkinson's disease with myasthenia gravis.

Key words: Parkinson's disease, myasthenia gravis, dysphagia, levodopa, dysarthria.

INTRODUCTION

Parkinson's disease is a neurodegenerative disorder characterized by tremors, rigidity, bradykinesia, and postural instability. Dysphagia and dysarthria are commonly seen in these patients (Felix et al 2008). 18-33% of patients with Parkinson's disease present with self reported dysphagia (Kalf et al 2012, Perez-Lioret et al 2012, Walker et al 2011). Dysphagia in Parkinson's disease may be apparent in any or all of the oral, pharyngeal, and esophageal stages of deglutition. Oral stage deficits occur most frequently in Parkinson's disease. It is usually the first indication of dysphagia in Parkinson's disease. Dysphagia can be made worse by lack of saliva or dry mouth in these patients. We report an interesting cause of dysphagia in a patient with Parkinson's disease.

Case Report

In October 2010 a 60 year old male patient presented M S Ramaiah hospital, Bangalore with progressive asymmetrical resting tremors of both upper limbs and slowness of activities. He was a known diabetic and hyper-

and was on medications. Neurological examination showed mask like face with decreased eye blinking. Cranial nerve examination was normal. On motor system examination, patient had rigidity and motor power was normal in all four limbs. Deep tendon reflexes were normal and plantar response was flexor. Patient had asymmetric resting tremors of both upper limbs (left > right). Magnetic resonance imaging of the brain performed at that time was normal. Hence diagnosis of Parkinson's disease was made and patient was started on levodopa-carbidopa (100mg/25mg) 1-1-1-1. Patient showed very good response to treatment and there was improvement in slowness of activities, rigidity, and resting tremors. The patient was not put on trihexyphenidyl.

On 24/10/2011 he presented with history of dysphagia, dysarthria, and diplopia of 6 weeks duration. He also gave history of nasal regurgitation. His neurological examination revealed bilateral ptosis, jaw weakness, and resting tremors of left upper limb. Lower cranial nerve examination was normal. Complete blood count, renal function tests, serum electrolytes, and liver function tests were within normal limits. Video laryngoscopy revealed pooling of saliva but there was no vocal cord palsy. Oesophagogastroduodenoscopy was done for evaluation of dysphagia, but it was also normal. Magnetic resonance imaging of the brain was also normal.

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As neuroimaging was normal and patient had bilateral ptosis and jaw weakness, neostigmine test was done to rule out myasthenia gravis and it was positive. Repetitive nerve stimulation test was positive for decremental response. Acetylcholine receptor antibody was positive and the titre was 19.60nmol/L (0 - 0.25nmol/L). Computed tomography of thorax was done to rule out thymoma, which was normal.

Based on clinical features and investigation findings patient was diagnosed to have myasthenia gravis. Patient was started on pyridostigmine and oral pednisolone. Within one week of initiating the above medications patient's symptoms improved substantially. Hence final diagnosis of Parkinson's disease with myasthenia gravis was made.

DISCUSSION

Dysphagia is commonly observed in patients with Parkinson's disease. Defects in all phases of swallowing have been documented in patients with Parkinson's disease (Johnston et al 1995). Whereas oropharyngeal weakness leading to dysphagia is rarely the presenting symptom of myasthenia gravis (Romo Gonzalez et al 2010 and Lanfranconi et al 2011). Myasthenia gravis is a disorder of neuromuscular junction characterized by weakness and fatiguability of skeletal muscles (Singhal et al 2008 and Wei et al 2003). Myasthenic patients have an increase incidence of several associated disorders like thymoma and autoimmune disorders. But occurrence of Parkinson's disease and myasthenia gravis in the same patient is rare.

Levin N et al (2003) reported a case series of 4 patients of Parkinson's disease with myasthenia gravis. The first patient had Parkinson's disease and 5 years later he developed weakness of neck extensors due to myasthenia gravis. The second patient presented with unilateral rigidity and tremors which was diagnosed as idiopathic Parkinson's disease and was started on levodopa. Four years later he developed intermittent ptosis, diplopia, difficulty in chewing and breathing. Patient had high serum acetylcholine receptor antibody titres. Patient's symptoms improved with pyridostigmine. The third and fourth patient were known case of myasthenia gravis and later developed Parkinson's disease.

Kao KP et al (1993) reported a case of 62 year old chinese female who had idiopathic Parkinson's disease for 8 years and myasthenia gravis since 1 year. Diagnosis of Parkinson's disease was made by presence of resting tremors, rigidity, bradykinesia and response to dopaminergic and anticholinergic medications. Diagnosis of myasthenia gravis was based on fluctuating ptosis and diplopia, and typical decremental response on repetitive nerve stimulation test. Treatment with pyridostigmine induced significant improvement.

Our patient was put on levodopa and we have not found

any literature showing that levodopa causes myasthenia gravis. Studies have shown that anti parkinsonian drugs like trihexyphenidyl can cause myasthenia gravis (Ueno et al 1987). But our patient was not put on trihexyphenidyl. Drugs like neostigmine used in treatment of myasthenia gravis might deteriorate the Parkinson's disease symptoms. Dysphagia is a common manifestation of advanced parkinson's disease. But in patients in whom dysphagia does not improve with levodopa like in our patient, one should keep possibilities of other etiologies for dysphagia in their mind and work up should be done to rule out other etiologies. So our patient having both parkinson's disease and myasthenia gravis might be coincidence and coexistence of both the diseases in same patient is very rare.

CONCLUSION

In a patient with Parkinson's disease, other causes like myasthenia gravis should be considered if the dysphagia is accompanied by other features that are unusual for Parkinson disease (like diplopia, facial weakness, ptosis, etc).

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