OPEN ACCESS Freely available online

Tremor and Other Hyperkinetic Movements

Case Reports

Hypothyroidism-induced Reversible Encephalopathy as a Cause of Aggravation of Parkinsonism and Myoclonus in Parkinson's Disease

Gwanhee Ehm[1], Han-Joon Kim[2,3,4] & Beomseok Jeon[2,3,4*]

[1] Department of Neurology, National Medical Center, Seoul, Korea, [2] Department of Neurology, College of Medicine, Seoul National University, Seoul, Korea, [3] Movement Disorder Center, Seoul National University Hospital, Seoul, Korea, [4] Parkinson Study Group, Neuroscience Research Institute, College of Medicine, Seoul National University, Seoul, Korea

Abstract

Background: Myoclonus and encephalopathy are unusual in patients with Parkinson's disease (PD).

Case report: We describe the case of a 59-year-old male with PD who developed myoclonus and encephalopathy. Underlying hypothyroidism was revealed after admission and treated with levothyroxine. Myoclonus and encephalopathy were completely resolved following thyroid hormone replacement.

Discussion: Hypothyroidism can cause reversible myoclonus and encephalopathy along with unusual aggravation of parkinsonism symptoms in patients with PD.

Keywords: Parkinson's disease, hypothyroidism, myoclonus, encephalopathy

Citation: Ehm G, Kim HJ, Jeon B. Hypothyroidism-induced reversible encephalopathy as a cause of aggravation of parkinsonism and myoclonus in Parkinson's disease. Tremor Other Hyperkinet Mov. 2017; 7. doi: 10.7916/D8BP0F9F

*To whom correspondence should be addressed. E-mail: brain@snu.ac.kr

Editor: Elan D. Louis, Yale University, USA

Received: August 16, 2017 **Accepted:** September 28, 2017 **Published:** October 19, 2017

Funding: None.

Financial Disclosures: None.

Conflicts of interest: The authors report no conflict of interest.

Ethics Statement: This study was reviewed by the authors' institutional ethics committee and was considered exempted from further review.

Introduction

Myoclonus is an unusual phenomenon in patients with Parkinson's disease (PD), especially non-demented patients. The development of myoclonus in the course of PD may herald alternative diagnoses such as corticobasal syndrome.[1] However, since myoclonus may be due to other medical conditions, a diagnosis other than neurodegenerative disease should be considered. Herein, we describe a patient with PD who had hypothyroidism presenting as abrupt development of myoclonus.

Case report

A 59-year-old male patient previously diagnosed with PD at age 48 was admitted for encephalopathy, severe exacerbation of parkinsonism, and myoclonus. He had been treated with levodopa and had an excellent response for the first 5 or 6 years of the disease. He underwent bilateral subthalamic nucleus deep brain stimulation surgery (DBS) at age 54 years because of severe dyskinesia and motor fluctuation despite meticulous medication adjustment. Following DBS, his parkinsonism was alleviated, and he could engage in socially active work. One month before admission, he had a delusion that a spy was watching him and visual hallucinations developed. His caregiver reported that the patient slept for almost 20 hours a day. His articulation deteriorated to the point where his family could hardly understand his words. He became unable to walk without assistance. His caregiver continued to regularly administer the antiparkinsonian drugs to prevent further symptom aggravation. Although the patient continued taking his usual PD medication, his condition worsened. On admission, generalized bradykinesia and rigidity were severely aggravated. Remarkably, myoclonus was present in all four extremities. The myoclonus appeared while maintaining posture and during action and was more prominent in both distal upper extremities, involving the shoulders. The mainly positive myoclonus was not stimulus sensitive. Electrophysiologic studies were not conducted. The results of thyroid function testing included in the routine admission blood work-up revealed an undetectable free thyroxine level and an elevated thyroid-stimulating hormone level of 200.6 μIU/mL (normal: 0.4-4.1 μIU/mL). Further investigation into the patient's medical history revealed

that he underwent total thyroidectomy for papillary thyroid cancer at age 56 and had been treated with levothyroxine at another hospital. However, he had accidentally stopped taking levothyroxine for about 2 months before the current admission. Based on his medical history and laboratory test results, levothyroxine therapy was restarted immediately. After supplementation, his parkinsonism, psychosis, sleep-related problems, emotional lability, and myoclonus markedly improved. By hospital day 10, his general condition stabilized to his previous disease status.

Discussion

Our patient showed rapid deterioration of psychotic, emotional, sleep-related, and motor symptoms over a few weeks, which is uncommon and may not be explained by exacerbation of PD itself. Myoclonus is also unusual in patients with PD. The prompt and dramatic improvement following levothyroxine administration further suggests hypothyroidism as the cause of this patient's rapid, global deterioration.

There is limited information on patients with aggravated parkinsonism due to hypothyroidism[2,3] or hyperthyroidism.[4,5] The accompanied encephalopathy and myoclonus, which were reversed by levothyroxine therapy, have not yet been reported in patients with PD and hypothyroidism. This lack of literature may be partly due to a low level of suspicion because the symptoms aggravated by hypothyroidism can be masked by and misconstrued as deterioration of PD itself.

Based on this didactic case and as pointed out by other authors,[2–4] thyroid function assessment should not be overlooked in patients with PD who experience rapid aggravation of parkinsonism, encephalopathy, and myoclonus. Since hypothyroidism can be managed with appropriate medication, it should be included in the list of differential diagnoses of unusual aggravation of parkinsonism and symptoms that are not typical for PD, especially when the patient has underlying thyroid disease.

References

1. Caviness JN. Myoclonus. *Parkinsonism Relat Disord.* 2007;13:S375–84.

2. Garcia-Moreno JM, Chacon-Pena J. Hypothyroidism and Parkinson's disease and the issue of diagnostic confusion. *Mov Disord.* 2003;18(9):1058–9. doi: 10.1002/mds.10475

3. Munhoz RP, Teive HA, Troiano AR, Hauck PR, Herdoiza Leiva MH, Graff H, et al. Parkinson's disease and thyroid dysfunction. *Parkinsonism Relat Disord.* 2004;10(6):381–3., doi: 10.1016/j.parkreldis.2004.03.008

4. Kim HT, Edwards MJ, Lakshmi Narsimhan R, Bhatia KP. Hyperthyroidism exaggerating parkinsonian tremor: a clinical lesson. *Parkinsonism Relat Disord.* 2005;11(5):331–2. doi: 10.1016/j.parkreldis.2005.01.009

5. Prakash KM, Kek PC. Hyperthyroidism "masked" the levodopa response in newly diagnosed Parkinson's disease patients. *Parkinsonism Relat Disord.* 2010; 16(10):691–2.