

Case Report

Severe Somatic Hallucination Induced by Antiparkinson Drugs: A Case of an Elderly Patient Misdiagnosed with Parkinson's Disease

Satoshi Ueda*

Department of Neuropsychiatry, Nippon Medical School, Japan

Corresponding author

Satoshi Ueda, Department of Neuropsychiatry, Nippon Medical School, 1-1-5 Sendagi, Bunkyo-ku, Tokyo 113-8603, Japan, Tel: 81-3-3822-2131; Fax: 81-3-5814-6287; Email: sat333@nms.ac.jp

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Abstract

Hallucinations are common as psychiatric symptoms in the treatment course of Parkinson's disease (PD). The majority of them are considered to be associated with both antiparkinson drugs and the disease process itself. Visual hallucination (VH) and delusion of persecution are very common, while olfactory or somatic hallucination (SH) is less common. The author reports an elderly female patient who exhibited severe SH accompanied by involuntary movement that disappeared after the discontinuation of all antiparkinson drugs. She had been misdiagnosed with PD. The present case might reveal "genuine" psychiatric side-effects induced by increasing numbers and doses of antiparkinson drugs. Interestingly, VH, which PD patients most often develop, was not observed here at all. It might be SH, not VH, that is more strongly associated with antiparkinson drugs. SH in this case consisted of two types of abnormalities in the sense of body balance and of being touched, and both have been rarely reported in other diseases. This case suggests that SH, although not very common in PD, may be a unique and noticeable symptom as the psychosis induced by antiparkinson drugs.

INTRODUCTION

Hallucinations are well known as psychiatric symptoms in the treatment course of Parkinson's disease (PD) [1,2]. The majority are associated with the increasing use and doses of antiparkinson drugs, while they are more related to the disease process itself in some cases. Visual hallucination (VH) is the most common, followed by auditory hallucination, and olfactory, tactile, and somatic hallucinations (SH) are also reported [3,4] although they are less common. The author describes an elderly female patient misdiagnosed with PD who exhibited severe SH that disappeared after the discontinuation of all antiparkinson drugs. This case may provide some suggestions concerning the manifestation of hallucinations in PD.

CASE PRESENTATION

The patient was a 74-year-old widow with no children. At the age of 63, she felt anxious about an increasing inability to cook and began to attend a psychiatric hospital with a diagnosis of depression, although details are unknown. Six years later, she was admitted to another psychiatric hospital due to relapse of depression and comorbidity of Parkinsonism including finger

tremor and freezing of gait. Her depressive state went into remission, and she could live her daily life with no help except for the use of a walking cane. At age 74, when she voluntarily entered a Nursing home, she was taking antiparkinson drugs containing combined levodopa 600mg/day and carbidopa 60mg/day, amantadine 300mg/day, and bromocriptine 15mg/day prescribed by a neurologist with the diagnosis of Parkinson's disease. She began to develop finger tremor and freezing of gait again, and selegiline and droxidopa were added by the neurologist who considered those symptoms an exacerbation of Parkinson's disease. Myoclonic movement of extremities was often observed, and she became non-ambulatory. Then combined levodopa and carbidopa were increased to 800mg/day and 80mg/day. Droxidopa was stopped. After a while, she was not even able to stand up due to severe tremor of extremities, pyrexia and body sweating. She also began to complain frequently, "I'm scared of falling!"

She was admitted to a psychiatric ward in a geriatric hospital on a stretcher. She showed high fever (39.2°C), tachycardia (110 bpm), high blood pressure (196/92 mmHg) and marked sweating in her whole body. She was alert and fully oriented. She constantly

thrashed her extremities about, although the medical staff tried holding them down, and she cried with a frightened face, "I'm scared! I'm falling! Feeling danger!" She could not ordinarily talk to others. Involuntary movement of extremities was coarse but quick, and appeared neither as feverish shivering, myoclonus, chorea nor athetosis. An attending neurologist identified her movement symptoms not as the condition corresponding to any specific neurological evaluation or diagnosis, but as a psychiatric condition. No muscle rigidity was observed except slightly in the cervix. Eye movement and deep tendon reflex were normal. Blood test including thyroid function revealed elevated data of white cell count ($11,840/\text{mm}^3$), creatine phosphokinase (286 IU/L) and sodium (150mEq/L). Brain MRI showed very mild cortical atrophy in frontal and temporal lobe and no vascular lesion, and electroencephalography showed no abnormal findings.

After intravenous haloperidol at 2.5mg was ineffective, intravenous diazepam at 5mg was administered, and both somatic hallucination with fright and involuntary movement became resolved in one minute or so. At the same time, body temperature, heart rate and blood pressure returned to normal levels. She was able to communicate with others, and take food and water with normal swallowing function. Because resting tremor or muscle rigidity was not observed at all, we considered her symptoms to have been induced by increased antiparkinson drugs, not those caused by PD. Selegiline, which was the most recently added drug, was stopped and amantadine was tapered. However, she still exhibited somatic hallucination and involuntary movement several times a day, both daytime and nighttime. Once this condition occurred, she always complained that her body was sinking down, leaning or being overturned, and often added that somebody was mounting on her body, pushing on her back or lifting up her legs, with nobody visible to her. Sweating and tachycardia accompanied these complaints. Because these symptoms did not improve in five or ten minutes, intravenous diazepam at 5 to 15mg resolved them. When the symptoms disappeared, she said, "I was afraid! I'm glad this is over!" All abnormal blood data became normal within a few days. Pheochromocytoma was suspected due to paroxysmal palpitation, but was ruled out after detailed examinations.

Amantadine was stopped on the 4th day after admission, and bromocriptine was also stopped on the 11th day. SH did not appear from 12th day and involuntary movement rarely occurred. On day 19, five days after combined levodopa and carbidopa were tapered to 500mg/day and 50mg/day, the movement disappeared completely. Manifestation or exacerbation of rigidity and tremor was also entirely absent, and her activity of daily living improved. Combined levodopa and carbidopa were tapered again, and were stopped on day 41. An attending neurologist's examination found no sign of Parkinson's disease. Cognitive function including recent memory or orientation was also intact. She was discharged on foot 50 days after admission, with no antiparkinson agents. For five years since discharge, she has had no neurological and psychiatric problems.

DISCUSSION

The present case is very rare in that many kinds of antiparkinson drugs were given to a normal subject. The severe SH and involuntary movement observed here proved not to be

related to PD at all. The treatment course and examinations clearly showed that the patient was not in delirium. The involuntary movement and autonomic symptoms might possibly suggest serotonergic syndrome induced by selegiline. However, the dramatic and rapid response to intravenous diazepam does not support this diagnosis, as there have been no reports showing such effectiveness of diazepam for the syndrome. With serotonergic syndrome, it would usually have taken much longer to clinically respond to any therapy than in this case. There have also been no reports of hallucinations in relation to the syndrome. Moreover, myoclonus and hyperreflexia are most typical of the syndrome, but they were absent here. The case was misdiagnosed as PD, and at maximum five kinds of antiparkinson drugs were administered. At the beginning, perhaps, the patient's Parkinsonism induced by antidepressants would have been falsely considered as that of PD. She was prescribed antiparkinson drugs, and then tremor and dyskinesia occurred. Although actually induced by antiparkinson drugs, those symptoms would have been considered exacerbations of PD, and those drugs were increased and added one after another. Probably five years had passed from the start of antiparkinson drugs to the development of somatic hallucinations.

Which antiparkinson drugs actually caused these symptoms could not be confirmed. Any one of the drugs might have caused them, or the combinations of two or three drugs might have done so. In any event, it is likely that the use of one or more drugs at a relatively high dose for a long period was strongly associated with development of the symptoms. In the treatment of this case, diazepam was very effective against involuntary movement, autonomic symptoms and somatic hallucinations. Diazepam, a benzodiazepine, has anti-anxiety, anticonvulsive, and hypnotic, but not anti-hallucinatory action. Therefore, it would not be easy to consider that diazepam had a direct effect on these symptoms. In this case, the symptoms were characterized by accompanying very strong fright. Fright may well be one of the target symptoms of diazepam. Eased fright may possibly have led to resolving involuntary movement and autonomic symptoms, then weakening somatic hallucinations, although its mechanism of favorable action could not be clearly explained.

This case might have shown "genuine" psychiatric side-effects induced by increased antiparkinson drugs. Psychotic symptoms in PD include mainly visual or auditory hallucinations and delusions of persecution [1,2], which are often difficult to treat only by pharmacotherapy [5]. It remains unclear whether these symptoms are related to the disease process itself (endogenous factors) or are induced by antiparkinson drugs (exogenous factors), although both factors have been considered to contribute more or less to their development [6]. Interestingly, the psychotic symptom in this case was not VH, which PD patients most often develop, but SH. Such involuntary movement and autonomic symptoms that accompanied SH also appear uncommon, although dyskinesia has been well known as a side-effect of the long-term treatment with levodopa. These facts suggest that the VH observed in many PD patients may be just slightly associated with antiparkinson drugs, whereas the SH observed in some patients may be more linked to those drugs.

With respect to symptomatology, SH in this case can be classified into two types of sense-related abnormalities; sense of body balance and of being touched. The abnormality in the sense of body balance includes the sense of one's body falling, leaning or being overturned, which are rare and unusual symptoms. These symptoms occur along with acute fright, accompanied by severe involuntary movement and autonomic change. These could be understood as an enhanced dopaminergic neural system by antiparkinson drugs. Although similar symptoms have been rarely known in other psychiatric disorders, there seems to be some similarity with "coenästhetische Schizophrenie (cenesthetic schizophrenia)", which Huber [7] proposed as the fourth (following paranoiac, hebephrenic and catatonic type) subtype of schizophrenia in 1957. Huber mentioned that this subtype exhibits a paroxysmal sense of falling or abnormal up-and-down movement, abnormal change of sweating, body temperature and sleep, or systemic coarse tremor that can be mistaken for feverish shivering. Interestingly, those signs closely resemble the symptoms of this case. Given the dopamine hypothesis of schizophrenia, antiparkinson drugs, which are dopaminergic, might well induce much the same symptoms.

The other type of abnormality is the sense of being touched such as invisible somebody's mounting on patients or pushing against their back. This symptom has been rarely reported in other diseases. It seems similar to tactile hallucination [4], but this is rather different from tactile hallucination in that it is the

perception of a larger body region being touched with more pressure to the body.

SH is not very common in the treatment course of PD. This case suggests, however, that it may be a unique and noticeable symptom as a psychosis induced by antiparkinson drugs.

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