Cenesthetic hallucinations in a patient with Parkinson's disease

Hallucinations are a side effect of treatment with levodopa and dopamine agonists. They are more common in patients with Parkinson's disease with advanced age and cognitive impairment.[1–3] Hallucinations secondary to dopaminergic drugs are usually visual, and less often, auditory.[4] We describe a patient who developed cenesthetic hallucinations during pergolide and levodopa treatment.

A 66 year old woman with Parkinson's disease, predominantly rigid akinetic, had been treated with carbidopa-levodopa since the age of 55 in 1984. When she was evaluated for the first time in our hospital in 1991, she was treated with 62.5/625 mg/day of carbidopa/levodopa divided into five doses, 5 mg/day selegiline, and 7.5 mg/day bromocriptine. She had motor fluctuations and mild peak dose dyskinesiae. Pergolide was introduced in gradually increasing doses up to 3 mg/day as replacement for bromocriptine, with a good initial response. However, in 1992, the parkinsonian symptoms had worsened progressively, and she spent around 60% of the day in "off" periods. Pergolide was increased up to 5 mg/day with a good response. In October 1993, standard levodopa was changed to a controlled release preparation of carbidopa/levodopa, up to 1400 mg/day divided into seven doses, and pergolide was reduced to 3 mg/day because of dyskinesiae. On this combination, the parkinsonian symptoms remained stabilised until July 1995, the "off" time being about 20% of the day. At that time, controlled release carbidopa/levodopa needed to be increased up to 450/1800 mg/day and pergolide up to 3.5 mg/day. In September 1995, the patient developed somatic hallucinations that she described as feeling as if her bowels and bladder extruded from the distal parts of her upper limbs. She tried to avoid the extrusion of these organs by compulsively scratching her arms, up to the point of inducing erosions. Reduction of pergolide to 2.5 mg/day and of controlled release carbidopa/levodopa to 350/1400 mg/day did not control the hallucinations, but they improved greatly with clozapine in gradually increasing doses up to 150 mg/day. The somatic hallucinations remained stabilised until November 1996. At that time the dose of clozapine needed to be increased up to 200 mg/day because of worsening. In January 1997, these symptoms are again well controlled.

Somatic hallucinations are defined as false sensations of things occurring in or to the body. When they are visceral in origin they are named cenesthetic hallucinations.[5] Our patient developed cenesthetic hallucinations that were likely to be related to the antiparkinsonian treatment and were controlled adequately with clozapine. To our knowledge, cenesthetic hallucinations had not been described in this situation previously, and should be added to the range of psychiatric side effects of antiparkinsonian drugs.

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1 Meco G, Bonifati V, Cusimano G, Fabrizio E, Vanacore N. Hallucinations in Parkinson disease: neuropsychological study. *Ital J Neurol Sci* 1990;**11**:373–9.
2 Friedman A, Sienkiewicz J. Psychotic complications of long-term levodopa treatment of Parkinson's disease. *Acta Neurol Scand* 1991; **84**:111–3.
3 Saint-Cyr JA, Taylor AE, Lang AE. Neuropsychological and psychiatric side effects in the treatment of Parkinson's disease. *Neurology*. 1993;**43**(suppl 6):S47–52.
4 Kippervasser S, Inzelberg R, Korczyn AD. Auditory hallucinations (AH) in Parkinson's disease (PD) [abstract]. IVth international congress of movement disorders. Vienna, Austria, June 17–21, 1996; poster 727. *Mov Disord* 1996;**11**(suppl 1):194.
5 Kaplan HI, Sadock BJ. *Comprehensive textbook of psychiatry*. 5th ed. Baltimore: Williams and Wilkins, 1989:474.

Neuroleptic malignant syndrome-like condition in multiple system atrophy

Neuroleptic malignant syndrome (NMS) usually occurs during treatment with neuroleptic drugs, but a similar condition may occur after a sudden withdrawal of antiparkinsonian drugs or an imbalance of the monoaminergic systems in the brain. On the other hand, extrapyramidal symptoms and dysautonomia are common in multiple system atrophy, which is a disorder of the monoaminergic system, affecting dopamine, noradrenaline, and serotonin. Catecholaminergic agents are sometimes used to treat multiple system atrophy, and NMS-like conditions may also occur in patients with multiple system atrophy.[1 2] We found six episodes of an NMS-like condition in three out of 14 patients with multiple system atrophy over a one year period (table).

Patient 1 was a 47 year old man who had been receiving antiparkinsonian drugs from the age of 45. Because the drugs did not cause much response, he needed an increasing dosage. On 12 October 1995, the daily dosage of bromocriptine was decreased from 26 mg to 8.6 mg and he was also given 6 mg trihexiphenidyl HCI.

After four days, he developed myalgia, hallucination, hyperhidrosis, and severe bradykinesia. On 19 October his body temperature was 37.7°C. He had high serum creatine kinase (5264 IU (normal <180 IU/l)) and was admitted.

Bromocriptine (26 mg/day) and dantrolene sodium produced an immediate response, but also urinary retention and orthostatic hypotension. Brain MRI suggested a diagnosis of multiple system atrophy. In February 1996, the addition of 50 mg/day trazodone HCI induced bradykinesia and rigidity. Serum creatine kinase was 2560 IU/l. Discontinuation of trazodone HCI and administration of 75 mg dantrolene sodium produced an improvement.

Patient 2 was a 58 year old man who had developed gait disturbance with parkinsonism at the age of 51 and was taking antiparkinsonian drugs. In July 1991, he received low temperature burning on his abdominal skin. After 10 days, he developed rigidity and was admitted to hospital. He had severe rigidity, pyrexia with high serum concentrations of creatine kinase (34 080 IU/l), blood urea nitrogen (51 mg/dl), and creatinine (10 mg/dl), urinary myoglobin (600 mg/day), and showed oliguria. He was diagnosed as having acute renal failure caused by myoglobinuria, and haemodialysis resulted in recovery. In January 1992, he was transferred to our hospital. In June 1993, he developed pneumonia and received antibiotics with a continuation of antiparkinsonian drugs. After recovery from pneumonia, he continued to have a pyrexia, increased rigidity, tremor, and bradykinesia. On 12 July, his temperature was 39.8°C with high serum creatine kinase (2418 IU/l). Disontinuation of L-threo-DOPS and an increase in bromocriptine (17.2 mg to 26 mg) with intravenous dantrolene sodium (40 mg/day) therapy produced improvement. Discontinuation of dantrolene led to a relapse. Increased dosage of levodopa/dopa-decarboxylase inhibitor (300/75 mg to 900/225 mg/day) produced a response. In August 1995, he had a body temperature of 40.3°C and raised creatine kinase (1200 IU/l) with no inflammation or altered medication. Treatment with intravenous dantrolene sodium (40 mg/day) induced recovery within three days.

Patient 3 was a 56 year old woman with a six year history of dysautonomia. At the age of 52, she was pyrexial in the summer. Four years later, she had severe ataxia with hypotonia and no involuntary movements. On 10 July 1987 L-threo-DOPS (600 mg/day) was added to the previous drugs to decrease

Description of patients

Patient	*Illness*	*Sex*	*Age*	*Duration of illness*	*Month of NMS-like episode*	*Maximum body temp (°C)*	*Maximum serum CK (IU/l)*	*Cause*	*Course*
1	SND	M	47	3	October	37.7	5264	Decrease of bromocriptine	Recovery by bromocriptine and dantrolene
			47	4	February	37.4	2560	Trazodone	Recovery by bromocriptine and withdraw oftrazodone
2	SND	M	54	3	July	>39.0	34081	Low temperature burning	Renal failure. Recovery by dialysis
			56	5	July	39.8	2518	Pneumonia	Recovery by antiparkinonian drugs and dantrolene
			58	7	August	40.3	1200	Pyrexia	Recovery by dantrolene
3	OPCA	F	56	6	July	42.0	1500	Pyrexia + L-threo-DOPS	Died of disseminated intravascular coagulation

SND=Striatonigral degeneration; OPCA=olivopontocerebellar atrophy; CK=creatine kinase.