

FACTITIOUS DISORDER MIMICKING ADDICTION TO LEVODOPA IN A PATIENT WITH ADVANCED PARKINSON'S DISEASE

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ABSTRACT - We report a 43-year-old woman with early-onset Parkinson's disease in whom neurological control was impaired by psychiatric co-morbidity including major depression and panic disorder. The patient also met criteria for factitious disorder that mimicked dopamine dysregulation syndrome resulting in severe clinical and social disability.

KEY WORDS: Parkinson's disease, factitious disorders, dopamine dysregulation syndrome.

Transtorno factício mimetizando adição a levodopa em paciente com doença de Parkinson avançada

RESUMO - Relatamos o caso de mulher de 43 anos de idade com doença de Parkinson de início precoce cujo controle neurológico foi significativamente afetado por co-morbidades psiquiátricas incluindo depressão maior e síndrome do pânico. A paciente também apresentou critérios para transtorno factício o qual mimetizava a síndrome de desregulação dopaminérgica, sendo responsável por significativa incapacidade clínica e social.

PALAVRAS-CHAVE: doença de Parkinson, transtorno factício, síndrome de desregulação dopaminérgica.

While Parkinson's disease (PD) is traditionally considered a movement disorder, there is increasing awareness that behavioral and psychiatric symptoms constitute an important source of disability. Several neuropsychiatric syndromes are observed in PD including depression, anxiety, sleep disorders, psychosis and dementia¹. More recently, attention has been drawn to the compulsive use of levodopa in PD^{2,3}. These syndromes may be related to the adjustment reaction to the presence of a chronic progressive illness, to the basic pathology of PD or to the effect of antiparkinsonian drugs⁴.

We report a woman with advanced PD whose anxiety, depression, factitious symptoms, including simulation of compulsive use of levodopa, resulted in significant disability.

CASE

A 43-year-old woman, divorced, retired from public service and former teacher was sent to the Movement Disorders Clinic of the Federal University of Minas Gerais

with a long-term history of PD associated with unpredictable motor fluctuations as well as severe hyperkinetic dyskinesias related to the intake of levodopa. At age 31 she developed bradykinesia and fatigue, followed by resting tremor. She was eventually diagnosed with PD and started on levodopa. Despite the use of several pharmacological strategies, including anticholinergic drugs, dopamine agonists and levodopa isolated or in different combinations, the patient stated that her parkinsonian syndrome, particularly bradykinesia and gait disorder, had undergone steady deterioration. More detailed questions about her medical history lead to vague and angry responses. At the moment of referral, according to her report, she was using levodopa/carbidopa 50/12.5 mg every four hours, but remained most of the time akinetic, restricted to bed. She described to experience "on" periods not longer than 30 minutes when she was able to walk and to perform activities of daily living unassisted despite the presence of high amplitude adventitious movements. She also complained of feelings of sadness and fatigue, and recurrent ideas of worthlessness and death since one year before the referral. The patient reported paroxysmic attacks of intense anxiety associated with

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fear of dying, palpitations, sweating and shortness of breath when she was alone. On clinical examination, the most important findings were emotional lability, Mini-Mental State Examination score of 28/30, asymmetric (right greater than left) parkinsonian syndrome characterized by resting tremor, bradykinesia, rigidity and gait impairment (UPDRS score of 132/176 when "off", stage 4 of Hoehn-Yahr).

Considering the severity of patient's symptoms and signs, we decided to admit her to the ward. Depressive symptoms abated two weeks after beginning nortriptyline 75 mg per day. She did not present panic attacks during hospitalization. Despite optimization of antiparkinsonian treatment with scheduled levodopa doses (levodopa/benserazide 200/50mg 3/3 h) and addition of the catechol-O-methyltransferase, tolcapone 100 mg TID, the complaints of akinesia remained unchangeable. She demanded constant attention from staff to ease her suffering and frequently requested more levodopa and analgesics for relieving multiple pain symptoms, including headache, back and leg pain. Examining the patient several times a day, we noted that she was always akinetic, exhibiting severe generalized dyskinesias when the assessments were performed after presumed levodopa intake. In contrast, other patients in the ward informed us that the patient was able of eating and going to the bathroom alone. She could leave her bed when interested, especially when not observed by staff. The patient was self-administering pills of levodopa found in her belongings. She was also throwing away medicines offered by the nurses.

We considered that the patient was simulating her akinetic state as the symptom was much more related to the presence of the medical staff than to the "off" periods. This impression was corroborated by the fact she was voluntarily manipulating her prescription. Offer of psychotherapeutic support was refused. After one-month stay, she left the hospital without improvement in her parkinsonian symptoms.

On out-patient follow-up, she has modified the prescription on her own and continued complaining of akinesia most part of the day and dyskinesias after levodopa. She also reported several brief hospitalizations in other services with motor and gastrointestinal symptoms related to levodopa. The patient denied abusive use of levodopa, reporting the maximum daily dose of 0.5 g. Despite the alleged severity of symptoms, the patient has been capable of living alone with a 16-year-old son and doing activities of daily living. The clinical examination remained almost unchanged, revealing a severe parkinsonian syndrome with resting tremor, bradykinesia, rigidity and gait impairment (UPDRS score of 120/176 when "off", stage 4 of Hoehn-Yahr).

DISCUSSION

Long-term levodopa therapy in PD is commonly associated with a series of motor complications,

which include diurnal fluctuations or the "on-off" phenomenon and dyskinesias. Recently, it was associated with the compulsive use of levodopa (or other dopamine replacement therapy) characterized by levodopa-seeking and taking behavior called by some authors "dopamine dysregulation syndrome"^{2,3}. This syndrome seems to be uncommon and may become evident only during periods of hospitalization when additional doses of levodopa are constantly requested or when exaggerated "off" states occur³. In our case, these features were found in a patient with early-onset PD marked by severe motor complications. The comorbid depression could be a predisposing factor³. However, the diagnosis of this syndrome seems unlikely as the patient herein reported has had discordant characteristics from other cases with levodopa-seeking and taking behavior: report of frequent intolerable effects with levodopa and denial of use of large daily doses of it. On the other hand, the typical patients with addiction to levodopa deny side-effects related to this drug which is characteristically taken in very large doses.

One alternative hypothesis to explain the exaggerated akinetic state presented by the patient as well the manipulation of the prescription, including the self-medication behavior, would be factitious disorder. The failure to respond as expected to usual therapy, the demanding style of the patient, requiring constant attention and medications, the intentional production of symptoms to maintain the sick role and to receive support, and the recurrent hospitalizations would support the diagnosis⁵⁻⁷. It would be reasonable for the patient to keep a sick role as she complained of loneliness and even reported panic attacks when alone. Moreover, she did not have any external incentive for assuming this behavior, what would suggest malingering, an important differential diagnosis of factitious disorder⁷.

Searching the literature, we did not find any case report of factitious disorder in the course of PD. There are just few case studies of psychogenic parkinsonism defined as parkinsonism resulting from a primary psychiatric disorder, probably examples of somatoform and factitious disorder^{8,9}. Interestingly all patients with psychogenic parkinsonism showed slowness of voluntary movements and most also complained of this as dominant symptom⁸.

The outcome of our patient was poor as expected by the presence of comorbidity of severe motor complications and psychiatric symptoms, including

depressive, anxiety and factitious symptoms, and refusal of psychiatric support.

Psychiatric symptoms often go unrecognized in PD although they are potentially treatable and may be important factor to the morbidity of the disease. Accurate diagnosis represents a clinical challenge since these symptoms may be confused with manifestations of PD¹. The compulsive use of levodopa is a new described syndrome associated with PD that is being increasingly recognized^{2,3}. This case-report indicates that factitious disorder may be a differential diagnosis of this condition.

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