

SSRI-Induced Parkinsonism May Be an Early Sign of Future Parkinson's Disease

Sir: In recent years, there have been several case reports of extrapyramidal symptoms as a result of treatment with selective serotonin reuptake inhibitors (SSRIs).¹⁻⁴ These symptoms include dystonia, akathisia, and parkinsonism. In most previously reported cases, the patients had preexisting brain diseases, but some movement disorders developed after initiation of SSRI treatment in both young healthy and vulnerable old patients.^{2,3,5} Furthermore, SSRIs might change the symptom profile of preexisting idiopathic Parkinson's disease.⁶ There are reports of deterioration of parkinsonian patients taking SSRIs.⁷ The literature on this subject was recently reviewed by Caley.⁸

We report a case of a geriatric patient with depression who developed reversible SSRI-associated parkinsonism and was later diagnosed as having Parkinson's disease.

Case report. Ms. A, a 67-year-old woman with no previous psychiatric history, presented with major depressive disorder 2 years prior to the time of this report. She complained of sadness, initial insomnia, anhedonia, and feelings of hopelessness and helplessness that had begun 2 months before she sought treatment. She had been taking amlodipine besylate, 5 mg/day p.o., for hypertension for 1 year up to the time she presented with depression. She was started on treatment with fluvoxamine, 50 mg b.i.d., which was gradually increased over a 2-week period to 100 mg b.i.d. After 6 days on this dose, Ms. A developed a pill-rolling tremor, masked facies, bradykinesia, and a festinating gait with tendency to retropulse. Her family reported that she had fallen once while on this dose. Results of a cranial magnetic resonance imaging (MRI) scan were normal. The fluvoxamine treatment was discontinued 18 days after it was initiated, and Ms. A began treatment with maprotiline, 75 mg/day. All signs of parkinsonism resolved within 2 weeks. Her depressive symptoms decreased gradually. She stopped taking her drug by herself after 6 months. The onset of parkinsonian symptoms after fluvoxamine initiation and their rapid resolution after discontinuation of fluvoxamine were thought to be a medication-induced syndrome.

After an 11-month symptom-free period, Ms. A recently came to our neurology department with pill-rolling tremor, asymmetrical cogwheel rigidity, and bradykinesia. She was diagnosed as having Parkinson's disease; no depressive symptom had been detected. Selegiline was chosen for treatment.

Serotonin modulates dopamine in basal ganglia by inhibiting its production and release.⁸ Thus, increase in serotonergic transmission may cause parkinsonian symptoms in geriatric patients who may have less dopamine available in their nigrostriatal tract. Furthermore, SSRIs may worsen the symptoms of preexisting Parkinson's disease or depressive symptoms of anhedonia and social isolation. To our knowledge, this is the first case report of a patient with reversible SSRI-associated parkinsonian symptoms who developed Parkinson's disease in 2 years' time. If this finding is confirmed by further

clinical observations, SSRI-induced parkinsonism may be an early sign of Parkinson's disease in vulnerable geriatric patients.

References

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