Unsuspected, surreptitious drug-induced parkinsonism

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Parkinsonian signs are a common side effect of neuroleptic therapy.[1,2] We observed three cases in which neuroleptics were secretly administered at home to unaware consorts.

Case reports. Patient 1 was a 34-year-old man whose medical history had been uneventful until the summer of 1981, when tremor of upper limbs occurred. Based on the observation of resting tremor, impaired digital dexterity, and masked face, a diagnosis of juvenile parkinsonism was made by a neurologist. The patient was treated with levodopa and amantadine in increasing doses. This therapy was ineffective, and he was admitted to our department in April 1983. Upon observation, hypomimia, slowness of movements, cogwheel rigidity, resting tremor, and akathisia were found. Brain CT and routine examinations for metabolic disorders were normal. The patient denied having taken neuroleptics. After 1 week of treatment with trihexyphenidyl and propanolol, in addition to amantadine and levodopa, the parkinsonian picture significantly improved. After discharge from the hospital, he was treated with anticholinergics and levodopa. One year later, he accused his wife of poisoning him with drops of sedatives in meals. After separating from his wife, the parkinsonian signs reversed. In May 1985, antiparkinsonian drugs were withdrawn. The patient currently has no neurologic abnormalities.

Patient 2 was a 37-year-old clerk who had no neurologic problem until November 1984, when he complained of motor abnormalities that had appeared 1 month before and had progressively worsened. He had not taken any medication for the last 2 years. Upon observation in the ward, he had akathisia, bradykinesia, diffuse cogwheel rigidity, resting tremor, and a parkinsonian gait. Examinations for metabolic disorders (including Wilson's disease) were normal. A diagnosis of parkinsonism was made, and the patient was treated with antiparkinsonian drugs. Upon discharge, after 3 weeks of therapy, he presented with only mild parkinsonian signs and was given a prescription of benzhexol and bromocriptine. In February 1985, parkinsonian signs worsened and the patient was readmitted to the ward. After treatment with levodopa, deprenyl, and orphenadrine, a significant improvement of his clinical picture occurred. In July 1986 the patient was readmitted again due to a sudden akinetic attack. His neurologic examination showed a severe parkinsonian state, with prominent akinesia and rigidity. A variety of biochemical assays were performed on blood and urine samples, which detected traces of haloperidol in the urine. The patient was informed of this finding and he was treated as having a drug-induced parkinsonism. Since he had not taken haloperidol purposefully, we asked him to look for such a drug at home. A few months after discharge from the hospital, he brought us a sample of his breakfast coffee, in which we were able to detect haloperidol. After being informed of this finding, the patient separated from his wife. He currently has no neurologic abnormalities and takes care of his daughter, who is now 17.

Patient 3 was a 43-year-old man living in the same town as patient 1. In August 1988, he suddenly complained of dizziness and slowing of movements and mentation. He received a diagnosis of depression and was treated with amitriptyline. In October 1988, he learned from patient 1 that his (patient 1's) former wife, with whom patient 3 was then living, had chronically poisoned him with neuroleptics. Patient 3 then contacted us and was admitted to our ward. He presented a clear parkinsonian picture, with tremor of the hands, masked face, cogwheel rigidity, loss of finger dexterity, and slowness of speech. Search of phenothiazines and butyrophenones in urines showed traces of haloperidol. During hospitalization, the parkinsonism gradually improved. The patient was discharged a week later. We did not see him again, but we learned from his family physician that he had completely recovered from parkinsonism.

Discussion. Environmental factors may play a role in the etiology of Parkinson's disease[3]; in these patients, causative factors were in the home. Two patients were poisoned by the same woman; all received haloperidol. We did not ascertain why these women dosed their consorts with tranquilizers. All of them used drops of haloperidol, whose trade name (Serenase) may suggest a mild sedative action. As far as we know, these women were never confronted.

All the patients improved after admittance to the hospital (the first and the third quite significantly so) and worsened when at home. In the first two patients, akathisia was associated with parkinsonism. Concealed home poisoning should be suspected when parkinsonian features respond well in the hospital but significantly worsen at home, particularly if akathisia is also present.

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