Letter

Withdrawal akinesia

Sir: Akinesia is an extrapyramidal side effect of neuroleptics. Akinetic apathy, diminished spontaneity characterised by few gestures, slow movements, and difficulty with initiating routine activities, can be mistaken for depression or for residual schizophrenic symptoms. The symptoms may be alleviated by a reduction or discontinuation of the neuroleptic, or by addition of an anti-Parkinsonian drug. However, it is not generally recognised that severe reactions may also follow upon reduction of the neuroleptic dosage in patients on long-term drug therapy. We describe a patient in whom akinesia probably was induced by a dosage reduction.

A 50-year-old mildly retarded man was transferred to our facility from a community hospital. He had a history of neurotic depression and had been treated with varying dosages of chlorpromazine and thioridazine for the last six years. His chief complaints were hypochondriacal and suicidal thoughts. His neuroleptic regimen had been maintained on thioridazine 100 mg daily for the previous 12 months. Two months before the transfer, chlorpromazine 150 mg per day was added to control his hypochondriasis. Admission medications included thioridazine 100 mg daily, chlorpromazine 50 mg three times a day and flurazepam 30 mg daily, as required. On examination, he showed no remarkable neurological abnormality other than laconic and slightly retarded psychomotor activities. No signs of psychotic behaviour were noted. His past clinical history was incomplete with little information to indicate his neuroleptic regimen. His psychotropic drugs were gradually reduced over an eight day period to chlorpromazine 50 mg twice daily. As the dosages were lowered, he began to be sleepy, and became very withdrawn with flat facial expressions. He sat quietly with fingers curled over the armrest, remaining in the same posture for hours until someone talked to him. Chlorpromazine was further reduced to 50 mg daily on the 12th day of his admission, but this made him more withdrawn, sad, and anhedonic.

Excessive salivation, rigid posture, and shuffling gait became the most prominent symptoms on the 16th day. A mild cogwheeling of his arms was also noted. He was lethargic and stayed in bed most of the time. No other cholinergic symptoms such as gastrointestinal disturbance, inomnia, restlessness or diaphoresis were noted. Benztropine 2 mg twice daily was added and chlorpromazine was reduced to 25 mg daily as required on the 18th day. His symptoms rapidly remitted within the next three days and all medications were discontinued one week later. The improvements were maintained without further psychotropic medications.

Akinesia can be a serious behavioural side effect of neuroleptic drugs. The behavioural symptoms are often confused with either residual schizophrenic or depressive signs and unwarranted drug therapy may follow.1 When other extrapyramidal signs are missing, discontinuation of the neuroleptic or addition of an anti-Parkinsonian drug may be the only way to ascertain the diagnosis. The side effect is thought to be due to the disturbances in dopamine-acetylcholine balance in the basal ganglia caused by a neuroleptic drug and the subsequent cholinergic overactivity upon discontinuation of the drug. Hence, when anti-Parkinsonian drugs are withdrawn at the same time as neuroleptics, withdrawal extrapyramidal symptoms become more prominent.2 3 Our patient had been treated with chlorpromazine and thioridazine, both known to possess relatively strong anticholinergic properties compared to other neuroleptics. Thus, discontinuation of thioridazine and reduction of chlorpromazine during the first week of his stay at our facility appeared to be responsible in exacerbating the akinesia rather than psychosis. Another clue in differentiating the akinesia in our case was his increased somnolent behaviour as the neuroleptic dosages were tapered. It has been suggested that a simple and fairly accurate correlate of akinesia is drowsiness.4 The behaviour changes occurring for the first two weeks following the dosage reduction almost gave us an impression the drugs were necessary. Fortunately, additional signs of extrapyramidal reactions appeared on the 15th day, so the decision to add benztropine was easier. Without close observation of his behaviour changes and careful interpretation, one might have reinstated the neuroleptic regimen. Rapid improvement of the patient's symptoms following benztropine therapy confirmed our diagnosis.

References


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