Absence of Parkinsonism Among Patients in Long-Term Neuroleptic Therapy Who Abuse Cocaine

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The study used the United Parkinson's Disease Scale to compare parkinsonian signs and symptoms among 19 patients in long-term neuroleptic therapy who had a history of cocaine abuse with those among 24 similar patients with no history of cocaine use. There was no significant difference between the two groups' scores. The results suggest that chronic cocaine abuse is not a risk factor for parkinsonism among subjects in long-term neuroleptic therapy. (*Psychiatric Services* 48:95–97, 1997)

Neuroleptic medications have been the mainstay of treatment for chronic schizophrenic illness since the 1950s. Depot neuroleptics, developed in the early 1980s, allowed a convenient once-a-month dosing schedule for patients with chronic schizophrenia. Depot neuroleptics have helped circumvent the problem of noncompliance with medication regimens among chronically ill patients.

Neuroleptics improve mental illness by putative dopamine receptor blockade. However, this mechanism also produces undesired side effects such as dystonic reactions among patients who receive acute treatment with neuroleptics and parkinsonism among patients in long-term neuroleptic therapy. The incidence of parkinsonian symptoms and signs in the population of chronic outpatients is estimated to be between 30 and 50 percent (1).

A significant number of patients with chronic schizophrenia abuse cocaine (2). Chronic abuse of cocaine leads to central dopamine deficiency (3). Because both neuroleptics and chronic cocaine use cause functional dopamine deficiency, it is reasonable to expect an aggravation of or an increase in parkinsonian signs among patients in long-term neuroleptic therapy who abuse cocaine. The study reported here tested this hypothesis.

Methods

The outpatient psychiatric clinic of the Veterans Affairs Medical Center in Philadelphia operates a depot neuroleptic program that serves 165 patients. The program is managed by a clinical nurse specialist, who administers injections of the medication and monitors the patients' mental status and general health.

The majority of the patients in the depot program have a history of long-term psychiatric hospitalizations. Most of the patients reliably keep their scheduled appointments for injections. All of the patients have taken oral neuroleptics for several years and continue to take them in addition to depot neuroleptics.

The study sample of 43 patients who agreed to participate were recruited over a period of two months. Patients had been receiving depot neuroleptics for a year or more. All 43 patients were male.

After the patients provided written consent to participate in the study, they were examined using the 14-item, 70-point United Parkinson's Disease Scale. The scale rates symptoms and signs and provides a score that indicates the presence or absence of parkinsonism through comparison with control subjects. All patients were examined by one of the authors, who was blind to patients' use of cocaine.

Cocaine use was difficult to identify due to the unreliability of patients' reports. Cocaine abuse was determined based on the patient's history and on problems listed in outpatient records. A urine test for cocaine metabolites was used to detect whether patients had used cocaine within the past two to three weeks.

Data on patients' age and the duration of therapy with depot and oral neuroleptics and anticholinergics were obtained from patients' records. The t test was used to compare the mean score on the United Parkinson's Disease Scale of patients with a history of cocaine abuse and that of patients with no history of cocaine use.

Results

All patients in the study were receiving either fluphenazine or haloperidol in the depot form. Data on the

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Table 1

Mean duration in years of neuroleptic and anticholinergic therapy among patients in long-term neuroleptic therapy, by whether patients have abused cocaine

Medication	Cocaine abuse (N=19)			No cocaine abuse (N=24)		
	Mean	SD	Range	Mean	SD	Range
Depot neuroleptics	4.1	2.3	1 to 10	6.4	4.9	1 to 22
Oral neuroleptics	18.6	5.7	2 to 27	22.0	8.4	3 to 39
Anticholinergics	15.4	8.5	0 to 27	18.0	10.6	0 to 39

patients' mean duration of therapy with oral and depot neuroleptics and with anticholinergics are shown in Table 1. There were no significant differences on these variables between patients with a history of cocaine abuse and those with no history of abuse.

Nineteen patients had a history of cocaine abuse. Their mean age was 42.5±6.5 years, with a range from 31 to 58 years. They had used cocaine for a mean±SD of 10.68±4.6 years, with a range from two to 18 years. The 24 patients with no history of cocaine use ranged in age from 34 to 67 years, with a mean of 47.7±9.4 years. Ten of the patients with a history of cocaine abuse, or 53 percent, had positive urine tests for cocaine metabolites, compared with none of the patients with no history of cocaine use. Clinical examination and behavioral observation indicated that the patients with a history of cocaine abuse were not cocaine intoxicated at the time of the examination for parkinsonian signs and symptoms.

The patients with a history of cocaine abuse had a mean score of .89±.66 on the United Parkinson's Disease Scale, with a range from 0 to 2. The patients with no history of cocaine use had a mean score of 1.5±1.4, with a range from 0 to 5. The difference between the two groups was not significant.

Discussion and conclusions

Comorbidity of substance use disorder and major psychiatric illness has been recognized as a serious problem (4), and a significant incidence of substance dependence has been found among psychiatric inpatients (5). For the outpatient clinic population represented in our study, an incidence of cocaine abuse of 21.7 percent has been reported (2). However, our study results suggest that chronic cocaine abuse may not be a risk factor for the development of parkinsonism among patients in long-term neuroleptic therapy.

Although the mechanism of cocaine-induced euphoria is not clear, it has been suggested that cocaine binds to the presynate transporter, thereby blocking the reuptake of dopamine, resulting in potentiation of neurotransmission (6). Cocaine may thus cause acute dopamine excess in the synaptic cleft, resulting in the euphoric effect. However, chronic use of cocaine eventually causes depletion of dopamine (3) and a decrease in the number of 3H-mazindol-labeled dopamine transporter receptors in the striatum (7). In a study of recently detoxified chronic cocaine abusers, postsynaptic dopamine receptors were found to be depleted (8).

It is well established that there is a loss of dopamine in the striatum of patients with idiopathic Parkinson's disease (9). The presence of dopamine deficiency in Parkinson's disease is clinically substantiated by the symptom improvement among patients with mild to moderate Parkinson's disease who receive dopamine replacement or dopamine agonists. Because dopamine deficiency is present in both chronic cocaine abuse and Parkinson's disease, it is reasonable to expect parkinsonian signs among chronic cocaine abusers. However, in another study we did not find parkinsonism among a group of chronic heavy cocaine abusers (10).

The occurrence of parkinsonism as a side effect of chronic neuroleptic therapy is thought to be due to chronic dopamine receptor blockage and to overactivity of the cholinergic system in the basal ganglia. Anticholinergic medication is commonly administered to counteract the side effects of neuroleptics. In this study, the majority of patients in both the cocaine-abusing and cocaine-free groups received anticholinergics. Therefore, the absence of parkinsonism among the patients who abused cocaine cannot be attributed to anticholinergic therapy.

In addition, the absence of parkinsonism among the cocaine-abusing patients in our study cannot be attributed to recent cocaine use. Acutely, cocaine blocks the reuptake of dopamine, causing an excess of dopamine in synaptic clefts. This process could theoretically improve parkinsonism. In our study, the patients with a history of cocaine abuse were not cocaine intoxicated at the time of the examination for parkinsonian symptoms, and only 53 percent of those subjects had positive urine tests for cocaine metabolites. In addition, the action of cocaine lasts only a few hours in the central nervous system.

Cocaine is thought to alter dopamine in both limbic areas and the basal ganglia. It is possible that the effect of cocaine on limbic dopamine is primarily responsible for the drug's euphoric effects. It is also possible that dopamine is quickly and adequately replenished in the brain. This mechanism may explain the absence of parkinsonism among the patients in the study.

Although the study findings suggest that cocaine use is not a risk factor for parkinsonism among patients in long-term neuroleptic therapy, patients receiving neuroleptics who abuse cocaine need interventions such as dual-diagnosis programs and support groups to increase their likelihood of remaining free of drug abuse. ♦

References

1. Grebb JK: Medication-induced movement disorders, in Comprehensive Textbook of Psychiatry, 6th ed. Edited by Kaplan H, Sadock B. Baltimore, William & Williams, 1995
2. Stone AM, Greenstein RA, Gamble G, et al: Cocaine use by schizophrenic outpatients who receive depot neuroleptic medication. Hospital and Community Psychiatry 44: 176–177, 1993

3. Dackis CA, Gold MS: New concepts in cocaine addiction: the dopamine depletion hypothesis. Neuroscience and Biobehavioral Review 9:469–477, 1985

4. Drake RE, Wallace MA: Substance abuse among the mentally ill. Hospital and Community Psychiatry 40:1041–1046, 1989

5. Miller F, Tanenbaum JH, Goodwin C, et al: Cocaine and polysubstance abuse in psychiatric inpatients. Hospital and Community Psychiatry 41:1251–1253, 1990

6. Cardoso F, Jankovic J: Movement disorders. Neurologic Clinics 11:625–639, 1993

7. Hurd YL, Harkenham M: Molecular alterations in the neostriatum of human cocaine addicts. Synapse 13:357–369, 1993

8. Volkow ND, Fowler JS, Wolf AP, et al. Effects of chronic cocaine abuse on postsynapse dopamine receptors. American Journal of Psychiatry 147:719–724, 1990

9. Kish SJ, Shannack K, Hoznykiewicz O: Uneven pattern of dopamine loss in striatum of patients with idiopathic Parkinson's disease. New England Journal of Medicine 318:876–880, 1988

10. Dhopesh V, Yagnik P, Weddington W: Can cocaine abuse cause parkinsonism? Presented at the Sixth Congress of the Pan-Arab Union of Neurological Sciences, Cairo, Mar 22–24, 1995

Clinicians' Predictions of Length of Psychotherapy

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The study examined therapists' accuracy in predicting the length of individual outpatient psychotherapy for 109 clients and attempted to identify variables associated with predicted and actual treatment lengths. The mean predicted treatment length (9.7 months) was significantly longer than the mean length of actual treatment (6.6 months). Therapists correctly predicted treatment length to the nearest month in 26 percent of the cases. Predictions were more accurate for older clients. Treatment tended to be shorter for clients with less than a high school education. Therapists more often predicted shorter treatments for clients with an adjustment disorder and those with less education. Predicting treatment length appears to be difficult. (*Psychiatric Services* 48:97–99, 1997)

Economic constraints and social changes have led to concerns among professionals and providers of funds about accurate treatment planning. Units of care, such as length of treatment, have crept into the psychotherapeutic vocabulary, and there is an increasing burden under managed care to operationalize treatment goals and to predict expected outcomes and expected treatment length (1). Many patients drop out early from treatment, which may indicate a need to include more precise considerations of length of treatment in planning.

Little research has been done on the accuracy of therapists' predictions of treatment length. In the few studies that have been reported, therapists' estimated treatment lengths were three times longer than actual treatment lengths (2,3) or were not significantly correlated with actual treatment lengths (4). In one study, client variables predicting treatment length were distance from the clinic and education (5). In another study, the variables were the client's forecasted treatment length, tendency to forget medical appointments, belief that others were responsible for the presenting problem, academic success, satisfaction that the therapist is listening objectively, and satisfaction with the professional status of the therapist (6).

The study reported here examined therapists' accuracy in predicting treatment length. It explored accuracy in predicting treatments of different lengths and identified variables related to therapists' predictions and treatment lengths.

Methods

The study included 109 consecutive first-time clients who were referred after intake for weekly individual psychotherapy over a one-and-a-half-year period at a community mental health center in central Israel. The clinic has an eclectic clinical orientation. There were no caps on treatment length, and treatment was paid for almost in full by the government.

Intake therapists were asked to pay special attention to completing the section of the clinic's standard treatment form that asks them to predict how many months treatment will last. Incomplete forms were returned to therapists for completion. Before the

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