A Survey of Tardive Dyskinesia in Psychiatric Outpatients

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The authors found a high prevalence (43.4%) of tardive dyskinesia in a sample of psychiatric outpatients, a population previously thought to be at nominal risk for development of this syndrome. There was no significant relationship between the presence of dyskinesia and age, sex, years of neuroleptic use, or various organic factors. The effects of dentures and of drug combinations are discussed, and it is noted that structured scales of dyskinesia and videotape recordings are important tools in diagnosing and following the course of dyskinesia.

TARDIVE DYSKINESIA, a neuroleptic-induced neurological syndrome, is characterized by involuntary movements of the tongue, face, jaws, and lips; occasionally, there are choreoathetoid movements of the extremities or trunk (1). Since the syndrome was first reported in 1956, various surveys have found its prevalence to be as high as 56%. These surveys focused exclusively on psychiatric inpatients, particularly chronic patients in geriatric, state, and veterans institutions (2, 3). The surveys have not included psychiatric outpatients, who are often younger, do not suffer the effects of chronic institutionalization, and have had shorter durations of treatment with antipsychotics.

Since tardive dyskinesia is often irreversible, it is essential that the treating physician be aware of its prevalence and of the factors that predispose to or in any way influence its occurrence. At present, the outpatient clinician does not have the necessary information to weigh this risk in treatment with antipsychotic medication. In recent years there have been reports of tardive dyskinesia in a few cases of younger patients who had been on neuroleptic medication for only brief periods of time (1). In this study, we surveyed a psychiatric outpatient population for tardive dyskinesia.

METHOD

Sixty-nine patients were randomly selected from the group of 250 patients receiving antipsychotic medication in the psychiatric outpatient clinic of Mount Sinai Hospital at the time of the study. Patients were included in the study, regardless of diagnosis, if they had been taking antipsychotic medication for at least 3 months and if they were not taking medication at the time of evaluation, but their last dose was within the 3 preceding months.

After giving informed consent, all patients were interviewed and examined by one of the investigators (G.M.A. or M.A.L.). The presence of dyskinetic movements and their location and severity were rated on the Abnormal Involuntary Movement Scale (AIMS), a 5-point scale that has been found to assess tardive dyskinesia reliably (4, 5). Videotapes were made of all patients who appeared to have involuntary movements and of 8 patients who seemed to have no such involuntary movements and who served as controls for the final evaluations. Three members of the research team (G.M.A., M.A.L., and R.C.D.), including an experienced neurologist who served as a consultant in developing the AIMS (R.C.D.), then made independent ratings of all videotapes by using the AIMS. For the purpose of the survey, patients were considered positive for dyskinesia only if all three raters concurred.

All patients were also evaluated for organicity by means of the Mental Status Questionnaire (MSQ) and the Face-Hand Test (FH) (6). The MSQ consists of 10 orientation and information questions and was rated positive if more than 2 incorrect responses were made. The FH test assesses the patient's ability to perceive double simultaneous stimulation; errors have been reliably associated with brain damage in adults. Patients were rated positive if they made 1 error or more.

Other factors evaluated for possible association with involuntary movements were the duration and type of medication, number and length of hospitalizations, history of amphetamine or alcohol abuse, history of

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ECT, personal or family history of neurological disorders, and history of drug-induced extrapyramidal side effects.

RESULTS

Of 69 patients sampled (53 women and 16 men), the three raters concurred that 30 patients (25 women and 5 men) were positive for dyskinesia on the AIMS. This represents 43.4% of our randomly selected outpatient sample. Although there was total agreement among raters on the presence or absence of dyskinesia, some variability existed in the severity of global ratings in 11 dyskinetic patients, with a grade of 1 being the largest difference between any two raters. Of the dyskinetic group, 60% were rated as minimally impaired, 30% as mildly impaired, and 10% as moderately impaired.

Demographic Characteristics

As can be seen in table 1, tardive dyskinesia was present in 47.2% of the women and 31.2% of the men surveyed, a difference that did not reach statistical significance (p=.40).

The mean age of the survey population was 45.5 years; the dyskinetic group had a mean age of 45.8 years and the nondyskinetic group of 45.3 years (p=n.s.). The percentage of patients diagnosed as dyskinetic was the same for those 50 years old or younger and those 51 or older (43.4%).

The mean age of the men with dyskinesia (37.8 years) was similar to that of those without dyskinesia (39 years). Mean ages for the two groups of women were also similar (47.4 years for the dyskinetic group and 47.8 for the nondyskinetic group). The sample is broken down by age and diagnosis of dyskinesia in table 1. The youngest patients with dyskinesia were 3 women aged 20, 22, and 28 years, and 1 man aged 20.

Medication and Organic Factors

The mean number of years on neuroleptics was slightly less in the dyskinetic group than in the nondyskinetic group (4.59 years versus 5.50 years, p=n.s.). It can be noted in table 2 that 46.7% of the patients with dyskinesia had been on neuroleptics for 2 years or less. Four patients (13.3%) with dyskinesia had received neuroleptics for 1 year or less, with the shortest duration being 8 months. All of the men with dyskinesia had been on neuroleptics for 5 years or less, the majority (4 of 5) for 2 years or less. Of the 30 dyskinetic patients, 2 had terminated neuroleptics 1 week prior to the evaluation. Both patients were immediately restarted on their medications, and 2 months later they were again found to be dyskinetic.

Despite careful assessment of hospital charts and interviews with patients, we were unable to assess with confidence all of the past neuroleptics and dosages patients had been given since their treatment began. Therefore, we could not assess which types of medications and dosage levels may be more often associated with tardive dyskinesia. The butyrophenones, phenothiazines, and thioxanthenes were all noted in the medication histories of patients with dyskinesia in this survey.

Neurological histories and history of ECT, alcoholism, amphetamine abuse, and cerebral dysfunction as reflected by the MSQ or FH test were approximately the same in the two groups (see table 3). Chart review revealed no active medical or neurological problems, no endocrinopathies, and no abnormal blood chemistries. Family history of movement disorders was found in 1 dyskinetic patient (Parkinson's disease) and

TABLE 1
Frequency Distribution By Age

	Dyskinetic Group (N=30)				Nondyskinetic Group (N=39)			
			Total				Total	
Age (years)	Women	Men	N	%	Women	Men	N	%
18–40	8	3	11	36.7	10	6	16	41.0
41–50	8	1	9	30.0	6	4	10	25.7
51–60	5	1	6	20.0	4	1	5	12.8
61–70	1	0	1	3.3	5	0	5	12.8
71–80	3	0	3	10.0	2	0	2	5.1
>80	0	0	0	0.0	1	0	1	2.6
Total	25	5	30	100.0	28	11	39	100.0

TABLE 2
Duration of Neuroleptic Medication

	Dyskinetic Group (N=30)				Nondyskinetic Group (N=39)			
			Total				Total	
Duration (years)	Women	Men	N	%	Women	Men	N	%
≤1	4	0	4	13.3	8	1	9	23.1
1–2	6	4	10	33.3	1	3	4	10.3
2–5	6	1	7	23.4	9	4	13	33.3
5–10	5	0	5	16.7	5	1	6	15.4
10–21	4	0	4	13.3	5	2	7	17.9
Total	25	5	30	100.0	28	11	39	100.0

TABLE 3
Organic Factors and Tardive Dyskinesia

	Dyskinetic Group (N=30)		Non-dyskinetic Group (N=39)	
Factors	N	%	N	%
Previous ECT	5	16.7	7	17.9
Alcoholism	6	20.0	7	17.9
Personal neurological history	2	6.7	2	5.1
Family neurological history	1	3.3	2	5.1
Amphetamine abuse	2	6.7	2	5.1
Positive MSQ or FH test	2	6.7	6	15.4

2 nondyskinetic patients (Parkinson's disease and familial tremor). No evidence of Huntington's chorea was present in patients or family members.

By chart review and interviews with patients, we verified past drug-induced dystonia in only 3 dyskinetic patients (10%) and 1 nondyskinetic patient (2.6%).

Miscellaneous Factors

The degree of psychopathology was assessed indirectly by the total number of psychiatric hospitalizations. The two groups had approximately the same mean number of hospitalizations (1.6 and 1.5). Medians were 2 and 1 for the dyskinetic and nondyskinetic groups, respectively. Statistical analysis indicates a trend toward more than 1 hospitalization among the dyskinetic group ($\chi^2=3.76$, $p<3.06$). Because patients' memories and charts were often vague about the duration of each hospitalization, exact times were not calculated, but all were for less than 1 year.

One factor that was frequently associated with dyskinesia was the presence of dentures—50% of patients with dyskinesia (N=15) had dentures compared with 18% (N=7) in the nondyskinetic group ($\chi^2=6.61$, $p=.01$). Of the 15 dyskinetic patients with dentures, 6 had vermicular movements of the tongue and 2 had choreoathetoid movements of the extremities. Only 6 of the dyskinetic patients with dentures had abnormal movements that were limited to their jaws.

An interesting finding was that in the total group of 69 patients surveyed, 9 patients were on neuroleptics alone and 5 of these (55.6%) had tardive dyskinesia. Of 22 patients on neuroleptics in combination with antiparkinsonians alone, 6 patients (27.3%) had tardive dyskinesia. Although a trend was noted, it was not statistically significant ($\chi^2=1.17$, $p=.28$).

DISCUSSION

Past surveys have reported the prevalence of tardive dyskinesia to range from 0.5% to 56%, with most recent studies citing the highest percentages (2, 3). These studies, which focused on chronically institutionalized populations, have given rise to the clinical impression that patients particularly at risk to develop dyskinesia 1) are female, 2) have organicity or prior brain damage (factors 1 and 2 are controversial), 3) are older (over 55 years old), and 4) have used neuroleptic medication for more than 2 years (1, 7, 8).

Before our outpatient survey of tardive dyskinesia, it was commonly felt that dyskinesia was infrequent in psychiatric outpatients being treated with neuroleptics and was mainly a problem of chronically institutionalized patients (1). The most striking result of our survey was the high prevalence (43.4%) of tardive dyskinesia in a population previously described as being at nominal risk for tardive dyskinesia. The dyskinetic group was young (average age of 45.5 years), had a short duration of neuroleptic medication (less than 2 years for 46.7%), and were not chronically hospitalized (median of 2 hospitalizations). There was no significant relationship between the presence of dyskinesia and age, sex, years of neuroleptic use, organicity, history of ECT, amphetamine or alcohol abuse, or of neurological disorders in the patients or their families. Perhaps the predisposing factors reported in previous studies were characteristic of their sample populations—chronically institutionalized patients.

Two associated findings of questionable significance were those related to dentures and antiparkinsonian medication. Dentures were worn by 50% of the dyskinetic group but only 18% of the nondyskinetic group, but only 6 dyskinetic patients who wore dentures had movements limited to the jaw, and none complained of discomfort. Since there were no denture problems and most of these patients had movements of the tongue and/or extremities, we believe that the movements were not induced by dentures.

Recently there have been reports that antiparkinsonians lower the threshold for amine stereotyped behavior in animals and when used in conjunction with neuroleptics may increase the risk of tardive dyskinesia in man (9). Our results suggested a contrary trend: 27.3% of patients on neuroleptics and antiparkinsonians had dyskinesia, whereas 55.6% of those on neuroleptic medication alone were dyskinetic. However, since our survey was not designed to evaluate the effects of antiparkinsonians specifically, it is difficult to draw conclusions from these data.

It appears that there is little to guide the clinician in predicting which outpatients may develop dyskinesia. We would agree with the present literature that the best way to reduce the occurrence is to give neuroleptic medication only to schizophrenic patients, using non-neuroleptics to treat other states, such as anxiety and depression. Since dyskinesia is often irreversible, it is incumbent on the physician to detect the syndrome early in its course so medication can be discontinued if the mental condition permits. Frequent examinations of the patient, assessing early oral-facial movements, particularly vermicular movements of the tongue, and temporary withdrawal of antipsychotic medication to evaluate an early dyskinesia that may have been masked have been suggested (10). We believe that early detection of dyskinesia is facilitated by using a structured dyskinesia scale like the AIMS and scales developed by Simpson and associates (11) and Crane and Naranjo (12), which can bring out subtle dyskinetic movements. This should be done before initiating treatment and at fixed intervals thereafter.

In the present study, the videotape recording of the dyskinesia examinations was invaluable in increasing diagnostic sensitivity, particularly of early signs of tardive dyskinesia. The videotapes included not only the examination itself but also a 1–2-minute interval of the patient sitting alone in the room. One patient did not show dyskinetic movements during the structured evaluation, but clearly had such movements during the period when he sat alone.

The videotape also allowed several observers to rate

the same examination at a later date. This is important since movement disorders can vary from one examination to the next in quantity and quality. The recordings can also be used to follow the development of these movements and to determine how they first appear or change over time. Most studies of tardive dyskinesia have not used videotape or movie recordings of the dyskinesia evaluations. Such recordings seem to be essential to future research on the clinical manifestations and progression of tardive dyskinesia.

CONCLUSIONS

This study demonstrates that outpatients as well as inpatients receiving neuroleptics are at a substantial risk for tardive dyskinesia. When neuroleptics are essential to the treatment of the patient, early detection of involuntary movements becomes critical. The present study suggests that the sensitivity of diagnosing tardive dyskinesia can be increased by examining the patient with a structured dyskinesia rating scale and videotaping the examination for evaluation by other physicians. Early, subtle signs can be detected more easily by following the patient's progress with subsequent videotaped evaluations. Further study is necessary to better understand the effects of medication combinations and other factors on the occurrence and development of tardive dyskinesia.

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