TARDIVE DYSKINESIA IN CHRONIC SCHIZOPHRENIC INPATIENTS

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SYNOPSIS

The prevalence of tardive dyskinesia in long-stay patients with schizophrenia in Woodbridge Hospital was found to be 2.5 per cent. The frequency increased with age and a preponderance observed in the age group between 55-59 years. There was no sex difference and patients with tardive dyskinesia did not differ significantly from a control group on the duration of neuroleptic treatment or total amount of anticholinergic drug prescribed.

INTRODUCTION

After the clinical application of Chlorpromazine in 1952, the occurrence of dyskinetic movements in the oral region believed to be associated with the use of the drug was recognised before the end of the decade. Later studies described other features of this syndrome which became known as tardive dyskinesia (1).

The cephalic triad of bucco-linguo-masticatory (BLM) movements is of central diagnostic importance. It also includes an axial and limb syndrome which may be a mixture of akathisia, athetosis and true choreiform movements but not rhythmic tremor. The onset is insidious and characteristically involves initially the tongue, where fine vermicular movement may be the first sign. This occurs on drug withdrawal or during treatment (2). Tardive dyskinesia interferes with eating, swallowing, respiration and social activities. It is aggravated by anxiety and diminished with drowsiness or when the affected muscles are involved in voluntary action.

In this present study we wanted to ascertain the prevalence of tardive dyskinesia in a population of hospitalised chronic schizophrenic patients. Secondly, we hoped to investigate how patients with tardive dyskinesia differed from a matched group of hospitalised patients without tardive dyskinesia in the duration of treatment with neuroleptics (chlorpromazine, thioridazine and trifluoperazine) and amount of anticholinergic drug (benzhexol).

MATERIAL AND METHODS

The epidemiological survey was conducted in March 1981 in the long-stay wards of Woodbridge Hospital. The dyskinetic movements were assessed on a rating scale devised by Simpson (3). Items on the scale refer to movements of the facial musculature, neck and trunk, and limbs. The time of the day for assessment was standardised since there is reason to predict a diurnal fluctuation in tardive dyskinesia (4).

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A control group present in the same wards as the dyskinetic patients but without apparent clinical manifestation was selected to match according to the following criteria: they were to be of the same sex, of a similar age (± 5 years) and should have been ill for a similar span of time (± 5 years). All the patients in the study were hospitalised for more than 5 years.

The total duration of neuroleptic drug treatment and amount of anticholinergic drug received by patients in both the experimental and control groups were calculated from the case records.

RESULTS

The prevalence of tardive dyskinesia was 2.5 per cent. The frequency for female patients was higher than male as presented in Table I. However this difference was not statistically significant.

TABLE I

Prevalence of tardive dyskinesia

	Total	Male	Female
Total number of patients	320	200	120
Patients with tardive dyskinesia	8	4	4
Per cent	2.5	2	3.3

x^2 (Yates correction) = 0.133, df 1, Not significant.

The number of patients with tardive dyskinesia increased with age in both sexes as shown in Table II. The youngest patient was 42 years and the oldest 64 years old. Most cases were found in the age group between 55–59 years.

TABLE II

Age distribution of patients with tardive dyskinesia

Age Group (Yrs)	Female	Male	Total
40 - 44	1	0	1
45 - 49	0	1	1
50 - 54	0	1	1
55 - 59	2	2	4
60 - 64	1	0	1

The most common presentation was bucco-linguo-masticatory movement and two female patients had dyskinesia involving both face and limbs.

TABLE III

Types of dyskinesia

	Male	Female	Total
Face	4	2	6
Limbs	0	0	0
Neck/trunk	0	0	0
Combination	0	2	2

In Table IV and V the characteristics of the experimental and control groups were compared. There was no significant difference between the two groups with regards to age and duration of neuroleptic treatment. The mean dose of anticholinergic drug received per month was not significantly different for both the group.

TABLE IV

Mean age and duration of treatment

	Age (Yrs)	Duration of treatment (Yrs)
Tardive dyskinesia	53.9	9.5
Control group	51.8	9.7
	t = 0.63 not significant	t = 0.18 not significant

TABLE V

Use of Anticholinergic drug (benzhexol)

	No	Yes	Total amount (mg/mth)
Tardive dyskinesia	0	8	167
Control group	1	7	155.5
	t = 1.02 not significant		

DISCUSSION

It is still controversial whether neuroleptic treatment per se can produce tardive dyskinesia (5). There may be other factors which could act together with neuroleptic drugs to give rise to dyskinetic movement. A contributing factor like brain damage can increase the vulnerability of the extrapyramidal system to dyskinesia (6). It is noted that in line with other observations (7), our study reveals that tardive dyskinesia increases with age and there is no sex difference.

The frequency of tardive dyskinesia among hospitalised patients varies in different studies between 0.5 per cent and 40 per cent (5). Our prevalence rate of 2.5 per cent is low. Smith et al (8) found no correlation between dyskinetic movement and dosage of neuroleptic drugs prescribed. In this survey the duration of neuroleptic treatment did not differentiate the experimental and control groups – similar finding was concluded by Perris et al (7).

The theory of counterbalancing dopaminergic-cholinergic transmitter system (9) for maintenance of normal movement suggests in tardive dyskinesia a relative dopaminergic hyperfunction or cholinergic hyperfunction. Administration of anticholinergic drug can therefore aggravate the dopaminergic-cholinergic disequilibrium produced by neuroleptic drugs. Our result did not show that dyskinetic patients were on a higher dose of anticholinergic drug compared with the control group.

Many remedies have been tried in tardive dyskinesia; the outcome is unimpressive. Increasing the dosage of the antipsychotic drugs only postpones risk since further and perhaps more severe dyskinesia may develop later. Aminedepleting drugs like reserpine seem only temporarily effective; the results with acetylcholine – increasing drugs such as cholin and deanol are unconvincing; the benzodiazepines and drugs acting on the gamma-amino-butyric acid

mechanisms provide only partial relief. Some of these apparently unpredictable responses may be due to tardive dyskinesia being a heterogenous group of disorders with a variety of mechanisms.

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