Tardive Dyskinesia

Patients' Lack of Awareness of Movement Disorder

ROBERTSON MACPHERSON and RACHEL COLLIS

Of 113 patients in long-stay wards of a psychiatric hospital, 43 had TD. Twenty-six of the 39 patients who consented to take part in the study were unaware of abnormal involuntary movements. These patients scored significantly lower on a short test of cognitive function than patients who were aware of such movements. The diagnosis of schizophrenia, particularly the 'defect' state with cognitive deficit and negative symptoms, was found to be associated with lack of awareness of TD.
British Journal of Psychiatry (1992), **160**, 110–112

Tardive dyskinesia (TD) is a disorder of abnormal involuntary choreo-athetoid movements associated with the use of neuroleptic drugs. The incidence of TD during long-term neuroleptic treatment is conservatively estimated at 20% (Gerlach & Casey, 1988). It is considered a public health problem because of its prevalence, potential persistence, treatment resistance, and the sometimes severely disabling nature of the involuntary movements (Munetz, 1985).

The lack of awareness of movement disorder among patients with TD is often striking, and is a well recognised clinical finding. Alexopoulos (1979) found that in a group of 18 out-patients suffering with TD, none had complained to their therapists of their symptoms, and eight were unaware of movement disorder. Wojcik *et al* (1980) found that 16 of 40 patients denied awareness of abnormal movements. DeVeaugh-Geiss (1979) quoted a figure of 10 unaware patients out of 15. Rosen *et al* (1982) reported that patients were more likely to be aware of peripheral dyskinesia than orofacial movements, and that awareness of movement disorder increased with greater severity of TD. Alexopoulos (1979) suggested that actively psychotic schizophrenics are less likely to complain of TD than those who are not actively delusional or hallucinating.

In this study we sought to establish whether lack of awareness of TD is related to factors such as diagnosis, duration of neuroleptic treatment and institutionalisation, severity of movement disorder, cognitive impairment, and positive or negative schizophrenic symptoms.

Method

All 113 in-patients on eight long-stay wards were screened for TD using the Abnormal Involuntary Movements Scale (AIMS; Guy, 1976). This is a widely used scale which rates the severity of abnormal movements of the mouth, face, neck, trunk and legs, and the overall severity of movement disorder. Only patients with a total score of more than five were included. All patients scored one or more on the global severity rating and scored at least moderately in one of the subcategories of the AIMS. Patients with any medical condition which could provide an explanation for movement disorder were excluded. The diagnostic criteria for TD used in this study were relatively stringent (and may have excluded some mild cases), but this was considered necessary to improve diagnostic reliability.

Patients were questioned in detail about their awareness of movement disorder, and the degree to which movement disorders disturbed them. Standard questions were read to patients, explained where necessary, and responses were recorded verbatim. Patients were considered to lack awareness of movement disorder only if there was true denial. If a psychotic or trivial explanation was given they were considered to be aware.

Medical case notes were examined to establish the duration of hospital stay and the number of years since neuroleptic medication had been started. All patients included in the study had a history of at least three months' cumulative neuroleptic exposure.

A full mental state examination was performed and the 10-item Roth Hopkins test (Blessed & Thompson, 1987) was used to assess cognitive functioning in each patient. This is an abbreviated version of the Mental State Test Score and is quick and well tolerated. Scores less than seven indicate moderate to severe cognitive impairment.

Information from case notes and the mental state examination was used to make an ICD-9 diagnosis (World Health Organization, 1978). Patients who were given a diagnosis of manic-depressive psychosis had no evidence of schizophrenic symptoms on mental state examination or in the case notes and had positive evidence of mood disorder. The schizophrenic patients were assessed further by means of the Positive and Negative Symptom Scale (PANSS; Kay *et al*, 1987), a validated and widely used scale.

Results

Of the 113 patients assessed, 45 were found to have movement disorder. Four patients refused to take part in the study and two patients with Huntington's chorea were excluded.

There were 21 men and 18 women in the study group (average age was 62.6 years, range 40–84 years). Thirteen of the patients were aware of movement disorder, 26 were unaware.

Table 1
Comparison of patient variables according to awareness/lack of awareness of TD

	Aware	Unaware
Schizophrenia	4	19
Manic-depressive psychosis	6	3
Other	3	4
No. of men	7	14
No. of women	6	12
Mean (s.d.) Roth Hopkins score	7.46 (2.8)	4.77 (3.5)
Mean (s.d.) age: years	59.2 (6.8)	60.1 (10.5)
Mean (s.d.) AIMS score	16.2 (4.1)	15.5 (7.4)
Mean (s.d.) years on neuroleptics	20.5 (10.9)	23.9 (7.3)
Mean (s.d.) years since admission	22.5 (18.0)	28.1 (11.9)

The ICD-9 diagnoses of patients were as follows: schizophrenia, 23; manic-depressive psychosis, 9; other, 7 (this includes diagnoses of personality disorder, epilepsy, and Korsakoff's psychosis).

Table 1 shows the relationship between various characteristics and awareness of movement disorder. Those patients with an ICD-9 diagnosis of manic-depressive psychosis were found to be significantly more likely to be aware of disorder than the schizophrenic group (χ^2, $P<0.01$).

There were no significant differences between aware and unaware patients with regard to age, sex, AIMS score, years on neuroleptic treatment, or years since admission. However, cognitive function, as assessed by the Roth Hopkins score, was significantly worse in the unaware patients than in those who were aware of their TD (4.77 v. 7.46; Mann-Whitney U test, $P=0.027$).

The variables were further analysed according to psychiatric diagnosis of schizophrenia or manic-depressive psychosis. The only significant difference was found to be the mean Roth Hopkins score. Patients with an ICD-9 diagnosis of manic-depressive psychosis had higher scores (8.2 v. 4.7; Mann-Whitney U test, $P<0.01$). There was no significant difference between the manic-depressive and schizophrenic patients with regard to age (63.9 v. 62.1 years respectively) or number of years on neuroleptic treatment (18.8 v. 24.3 years respectively).

Within the group of schizophrenic patients the aware/unaware subgroups were compared according to their scores for positive and negative symptoms on the PANSS. Negative symptom scores were higher in the unaware group (mean score of 28.3 as compared with 21.7 in the aware group), but this difference did not achieve statistical significance (Mann-Whitney U test, $P<0.1$). Average positive symptom scores were similar in the subgroups of aware and unaware schizophrenics (mean score of 20.0 as compared with 17.3 in the unaware group).

There was a trend for the schizophrenic patients who were aware of TD to obtain higher Roth Hopkins scores than the unaware schizophrenics, but this failed to achieve statistical significance (mean Roth Hopkins score 6.75 (s.d. 2.2) in aware schizophrenics, mean score 4.26 (s.d. 3.3) in unaware group; Mann-Whitney U test, $P<0.1$).

Discussion

We found a point prevalence of 38% of TD in our sample, which comprised long-stay patients. This rate is comparable with those found in similar populations (Gerlach & Casey, 1988). That 26 of our study group of 39 patients with TD were unaware of their movement disorder supports our clinical impression and previous research findings that lack of awareness is a common feature of the syndrome (DeVeaugh-Geiss, 1979; Alexopoulos, 1979; Wojcik, 1980).

In our population patients with a diagnosis of manic-depressive psychosis were significantly more likely to be aware of their movement disorder than those with a diagnosis of schizophrenia. We also found that the lack of awareness of TD correlated with impaired cognitive function. The lack of awareness may thus be due to the cognitive impairment associated with chronic schizophrenia. Within the schizophrenic group, there were trends for lack of awareness of TD to be associated with both cognitive impairment and negative symptoms.

Alexopoulos (1979) discussed the possible reasons for lack of awareness of TD. He suggested that in his group of 18 patients with TD, those schizophrenic patients who were unaware were more likely to be actively psychotic. However, we found that lack of awareness of TD did not relate to positive symptoms.

We found no correlation between awareness of TD and length of stay in hospital, duration of antipsychotic treatment, severity of TD, age, or sex. Thus, lack of awareness of TD is most likely to be a facet of the primary illness which leads to neuroleptic prescription, rather than the effects of neuroleptic treatment, TD itself, or institutionalisation. Other authors (e.g. Rosen *et al*, 1982) have reported that the degree of self-awareness of TD correlates with severity of movement disorder. We found no such relationship, although this may in part be due to the exclusion of mild cases.

The finding that large numbers of patients with TD do not complain of movement disorder, and that when asked many deny awareness of it, is important. Greater recognition of this among clinicians may encourage more active clinical assessments of patients at risk. Early detection of TD allows for more effective therapeutic intervention, and lessens the likelihood of severe irreversible movement disorder (Gerlach & Casey, 1988). Munetz & Roth (1985) found that patients who deny abnormal movements gain significant new knowledge from an informed consent procedure, but, interestingly, still continue to deny awareness of abnormal movements (10 out of 12 deniers on follow-up).

Access to this population of patients, in which an unusually large number of non-schizophrenic patients have been treated with long-term neuroleptic drugs, has given us an unusual opportunity to investigate the determinants of lack of awareness of TD. We have been able to demonstrate that the diagnosis of schizophrenia, particularly the 'defect' state, with cognitive deficit and negative symptoms, is associated with lack of awareness of TD.

Acknowledgements

The authors would like to thank Dr Malcolm Peet for his help and advice in the preparation of this paper. We would also like to thank Sharon Vaughan for producing the manuscript.

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R. J. Collis, MB, BS, MRCPsych, *Registrar in Psychiatry, Northern General Psychiatric Unit, Northern General Hospital, Sheffield*; *R. Macpherson, MB, ChB, MRCPsych, *Senior Registrar in Psychiatry, Ham Green Hospital, Pill, Bristol BS20 0HW*

*Correspondence

Cosegregation of Christmas Disease and Major Affective Disorder in a Pedigree

MICHAEL GILL, DAVID CASTLE and CONOR DUGGAN

Three males with factor-IX deficiency (Christmas disease) in one pedigree all had severe affective disorder. This apparent cosegregation, if true, would support the hypothesis that in some pedigrees, a gene for major affective disorder is located on the X chromosome.
British Journal of Psychiatry (1992), **160**, 112–114

Major affective disorder/manic depression is known to have a strong genetic component (McGuffin, 1988). Many studies have suggested an X-linked mode of inheritance in a proportion of families (Risch *et al*, 1986). Linkage studies have given attention to the q27–q28 region of the X chromosome, which includes the genes for protan and deutan colour blindness, glucose-6-phosphate dehydrogenase, and factor IX. Some studies have found evidence for linkage between X-chromosome markers from this region and manic depression (Mendlewicz & Fleiss, 1974; Baron, 1977; Mendelwicz *et al*, 1979; Baron *et al*, 1987; Mendlewicz *et al*, 1987). Others (Gershon *et al*, 1979; Berrettini *et al*, 1990) have failed to replicate the finding of X linkage. There is strong evidence of linkage to colour blindness in one set of pedigrees (Baron *et al*, 1987) and less robust evidence of linkage to the factor-IX locus in another (Mendlewicz *et al*, 1987). Although physically close, there is considerable genetic distance between these two markers which flank the fragile-X site. Linkage to both markers in the same pedigree is, therefore, unlikely unless there are two separate genes for major affective disorder.

We report a family in which three males with factor-IX deficiency (Christmas disease) all had severe affective disorders. The association, if true, would support the proposal that, in some families, a gene for affective disorder is located at the