Tardive Dysmentia: A Reappraisal

by Sukdeb Mukherjee

Abstract

The proposed concept of tardive dysmentia is reassessed with critical comments on methodological issues. The probability that this type of behavior is related to the disease process in chronic schizophrenia has not been ruled out. If tardive dysmentia were to be a result of neuroleptic exposure primarily, it should also be seen in neuroleptic-treated nonschizophrenic patients. In a study of tardive dyskinesia among bipolar patients, we failed to notice any cases that could have been described as tardive dysmentia as proposed. This suggests a strong possibility of this syndrome being illness related rather than neuroleptic caused. Finally, the syndrome appears to be more of a dyscontrol than a dysmentia. Tardive dyscontrol may be a better term than dysmentia. There is no evidence that these patients would fit criteria for a diagnosis of mania, and the proposal that these patients may be mistakenly diagnosed as manic is unfounded.

The article by Wilson et al. (1983) proposed the concept of tardive dysmentia and attributed the etiogenesis of this syndrome to long-term exposure to neuroleptics, postulating that the changing natural history of schizophrenia is to an important extent dependent upon the neurotoxic effects of chronic neuroleptic medication. They have reported significant correlations between the “severity of tardive dyskinesia” and some of their arbitrarily predefined tests for tardive dysmentia (unstable mood, loud speech, and euphoria). Interesting though the proposal may be, a number of methodological issues need to be pointed out:

• The authors have used the sum of the individual Abnormal Involuntary Movement Scale (AIMS) body scores to determine the severity of tardive dyskinesia (TD). This method is unsatisfactory as it would give the same total score to a patient with scores of “1” (minimal) on three body areas as to one with “3” (moderate) on one body area. Whereas the former does not meet research criteria for TD (Schooler and Kane 1982), the latter might. A better approach would have been to square the individual body area scores before summing them up as has been recommended by Murray Alpert (personal communication). Also, the research diagnostic criteria for TD (Schooler and Kane 1982) should have been used to demarcate between those with TD and those without.

• From the data presented, it appears that at least 50 percent of their patients would have met the severity criteria for a research diagnosis of TD. This is a somewhat high prevalence figure and may be suggestive of a sampling bias. In that case, caution should be exercised in generalizing from data obtained from this sample.

• The reason for inclusion of a single bipolar, manic patient is difficult to understand.

• The items used by the authors to assess tardive dysmentia are more suggestive of dyscontrol than dysmentia. Euphoria, excessive approach to the examiner, loud

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speech, instability of mood, and excessive words all imply a disinhibition of impulses and affective processes. The term dysmentia does not seem quite as applicable as dyscontrol or disinhibition. The probability that these symptoms are reflecting a illness-related syndrome rather than a neuroleptic-related syndrome has not been ruled out. This might be difficult to do as chronic schizophrenic patients who have not been exposed to long-term neuroleptic treatment are virtually impossible to find. On the other hand, there are groups of patients with long-term neuroleptic exposure in whom the natural course of the psychiatric illness does not suggest a dysmentia/dyscontrol syndrome developing. An example would be the case of bipolar disorder. We have studied TD in a cohort of 131 bipolar patients (Mukherjee et al., submitted for publication). The occurrence of TD was limited to the patients with long-term neuroleptic exposure history. However, none of these patients showed the type of behavioral picture proposed as being characteristic of tardive dysmentia during periods of remission from affective episodes. This does not support tardive dysmentia (dyscontrol?) as being a neuroleptic-induced neurotoxic phenomenon. However, it does not rule it out either. It may quite well be that certain schizophrenic patients do indeed develop this syndrome as a result of neuroleptic exposure to a brain which is dysfunctional in other ways. In other words, both the disease process and the neuroleptic exposure may be necessary, either one alone being insufficient. I have seen a number of cases in the long-term wards of state psychiatric centers who fit the description of the syndrome of tardive dysmentia.

- Finally, I find the authors' speculation on "pseudo-manic depression" being mistakenly diagnosed as "true" manic depression particularly disconcerting. Nowhere in their study have they provided evidence to support this statement. The characteristic symptoms of mania such as hyperactivity, decreased need for sleep, pressured speech, flight of ideas, grandiosity, racing thoughts, or hyperactivity are not reported to be found in excess in patients with tardive dysmentia. In an American psychiatric practice setting, with a long tradition of favoring a diagnosis of schizophrenia over mania in psychotic patients, the authors are presenting an unsupported argument to caution against the rediagnosing of certain "schizophrenias" to bipolar disorder. This is even more unjustifiable in the light of their proposing a new neurotoxic iatrogenic syndrome as a result of treatment with drugs that are primarily used in the pharmacotherapy of schizophrenia.

While bold speculations open the way for new discoveries, these should be done with great caution and bearing in mind the evidence. The concept of tardive dysmentia—at present merely an interesting speculation—needs to be observed more carefully. Unlike the skeptics who denied the possibility of neuroleptic-related tardive dyskinesia in the early years following the first reports of its occurrence, I would urge careful and systematic inquiry into the nature of this syndrome and its putative relationship with neuroleptic exposure. We have just concluded a study of this proposed syndrome in 60 inpatients and will be reporting on our findings in the near future.

References


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The Author Replies:

Mukherjee (1984) has raised a number of questions concerning our recent article "Is There a Tardive Dysmentia?" (Wilson et al. 1983). I would like to respond to several of his points and make some general comments as well.

In regard to our methods of diagnosis and assessment of tardive dyskinesia (TD), we were guided by the literature at the time the study was completed in 1978. The more recent diagnostic criteria and means of interpreting rating scales Dr. Mukherjee refers to should further enhance research in TD.

Concerning the relatively high