Prevention and Management of Tardive Dyskinesia

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Because no adequate therapy is available, the prevention of tardive dyskinesia is of great importance. The disorder may be prevented by periodic evaluations of the neurologic status of patients on long-term drug treatment, followed by reducing the drug dosage or discontinuing administration. Withdrawing neuroleptics, particularly in patients with pseudoparkinsonism, may uncover latent dyskinesia. For patients already exhibiting this disorder, the use of small doses of neuroleptics may be safe, provided changes in neurological symptoms are monitored carefully.

THE LITERATURE on tardive dyskinesia includes approximately 100 publications, and recent efforts to find a treatment for this condition attest to the growing concern on the part of psychiatrists. No paper, however, provides useful guidelines for the prevention and control of this very common and sometimes disabling complication of neuroleptic treatment.

In order to deal with this problem effectively, one must keep in mind the following facts:

1. In most instances the syndrome is clinically well defined and is different from pseudoparkinsonism or akathisia. Occasionally it resembles other neurologic diseases (1).

2. There is great variation in individual susceptibility, but prolonged exposure to drugs, use of high doses, and being older than 50 years of age predispose to this complication (2).

3. Pseudoparkinsonism may progress to a persistent disorder of a hyperkinetic type (3).

4. Tardive dyskinesia may not be evident while patients are receiving medication (4).

5. After drugs are withdrawn, symptoms may become more intense but may subsequently decrease or even disappear after several months off drugs (5).

6. Even though tardive dyskinesia is also known as persistent dyskinesia, my experience suggests that the earlier the symptoms are recognized and drugs are withdrawn, the better is the prognosis for a partial recovery.

7. Patients who exhibit tardive dyskinesia and who are maintained on relatively small doses of neuroleptics usually show little aggravation of symptoms over a one-year period (3).

8. The use of several drugs for the treatment of dyskinesia has proven unsatisfactory (e.g., pyridoxine and amantadine) or has not been tested adequately (e.g., tetrabenazine, reserpine, and L-methyldopa) (3).

9. While readministering neuroleptics to patients no longer on medication or increasing the dosage in patients receiving drug therapy may mask symptoms, they may at the same time cause further damage to the central nervous system (3).

Recommendations

In view of all this, I would like to make some recommendations. Patients receiving neuroleptics, particularly those on long-term drug therapy, should be examined at least once every three months for dyskinesia or for other types of neurological symptoms. The areas most likely to show motor abnormalities are, in order of decreasing frequency, the tongue, the fingers, the pelvis, the ankles, and the toes. About twice a year drugs should be completely withdrawn to unmask latent symptoms. This is particularly important for subjects with incipient manifestations or parkinsonian features. To avoid severe postural imbalance and/or disturbing oral dyskinesia, doses may have to be gradually reduced. The clinician will find that some 30 percent of chronic schizophrenic patients can

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maintain a satisfactory level of behavioral adjustment if they do not receive drugs for six months or longer (6). According to my observations, an additional 30 or 40 percent will regress mentally but will return to their base level within a few weeks when they are put back on small amounts of neuroleptics (50 to 300 mg. of chlorpromazine or equivalent doses of other drugs).

The use of higher doses may be necessary for severe psychoses. Such doses are safe for patients who have no evidence of tardive dyskinesia or who have only clinically insignificant manifestations. Parkinsonism, on the other hand, should not be allowed to persist for any length of time. Antiparkinsonian drugs may reduce overt symptoms of parkinsonism but may cause a shift to hyperkinesia (3).

The next question is: What can we do with the great mass of chronic patients already afflicted by tardive dyskinesia? The use of relatively small doses of neuroleptics entails little risk provided the clinician constantly monitors changes in the patient's neurologic status. In some cases of severe mental disorder, however, the prescription of a relatively large amount of medication is required, even in the presence of pronounced neurologic impairment. The danger of aggravating the situation may be very real, but a clear understanding of the total clinical picture will put the clinician in a position to weigh on an individualized basis the advantages as opposed to the disadvantages of drug therapy.

Conclusions

That so many patients suffer from tardive dyskinesia is regrettable. On the other hand, most subjects so afflicted experience relatively little discomfort. Furthermore, the grotesque facial grimaces, smacking of the lips, and bizarre activity of limbs and body are relatively minor problems for a chronic mental patient who spends his life in the sheltered environment of his home or of an institution. Consequently, an effort to suppress dyskinesia by reinstituting drug therapy, increasing doses, or shifting to other neuroleptic agents is definitely an unsound medical practice. Attempts to cover up overt neurologic manifestations may aggravate the syndrome in the long run or prevent the partial recovery one could expect from a drug-free period.

In my opinion, the readministration or use of higher doses of drugs should be considered only for the control of certain complications of tardive dyskinesia, such as ulcerations of the tongue or oral infections, or for the prevention of fractures and other injuries due to postural imbalance.

If these simple rules are followed, tardive dyskinesia will become a minor and rare complication.

REFERENCES

1. Crane GE, Naranjo ER: Motor disorders induced by neuroleptics. Arch Gen Psychiat 24:179-184, 1971
2. Crane GE: Neuroleptics and proneness to motor disorders. Read at the 124th annual meeting of the American Psychiatric Association, Washington, DC, May 3-7, 1971
3. Crane GE: Persistent dyskinesia. Brit J Psychiat (in press)
4. Crane GE: Tardive dyskinesia in patients treated with major neuroleptics: a review of the literature. Amer J Psychiat 124(Feb suppl):40-48, 1968
5. Degkwitz R: Extrapyramidal motor disorders following long-term treatment with neuroleptic drugs, in Psychotropic Drugs and Dysfunctions of the Basal Ganglia. Public Health Service publication no 1938. Edited by Crane GE, Gardner R Jr. Washington, DC, US Government Printing Office, 1969, pp 22-25
6. Prien RF, Levine J, Cole JO: High dose trifluoperazine therapy in chronic schizophrenia. Amer J Psychiat 126:305-313, 1969