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LETTER TO THE EDITOR

Tardive dyskinesia caused by the atypical antipsychotic risperidone and cured by the use of another drug of the same class, olanzapine

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Tardive dyskinesia, characterized by abnormal, involuntary, irregular choreo-athetoid movements of the muscles of the head, limbs, and trunk is a potential complication of antipsychotic drug therapy, especially with the classical antipsychotic medications. Although atypical antipsychotics have been introduced to cause no tardive dyskinesia there are some case reports of TD due to atypical antipsychotic drug use [1, 4]. Beyond these recent reports it has also been suggested that these novel agents may be beneficial in managing at least some types of dyskinesias [2, 3].

We would like to share the case report of a schizophrenic patient who developed severe dyskinetic movements 9 months after beginning treatment with risperidone and who responded to the use of another atypical antipsychotic, olanzapine, in a very short time.

Mr. T, a 29-year-old unemployed single man, was admitted to the psychiatric clinic of Akdeniz University with auditory hallucinations, delusions of persecutory type, lack of interest in nearly all daily activities and suicidal ideation. Interview and history-gathering revealed a period of 10 years characterized by predominantly negative symptoms of schizophrenia and two suicide attempts with only transient short-term antipsychotic and antidepressive medications. Fulfilling DSM-IV criteria for schizophrenia, the patient was put on risperidone, titrated up to the maintenance dose of 6 mg per day. Four weeks after starting antipsychotic therapy his baseline BPRS total score of 60 exhibited a decrement of 40%. Likewise, original SANS and SAPS scores of 70 and 35 displayed a remarkable reduction with scores of 40 and 15 respectively.

No problem emerged until the ninth month and the patient had nearly completely returned to his premorbid functioning level. But in his last examination he was found to be severly incapacitated by involuntary movements in his tongue, lips and face. The Abnormal Involuntary Movement Scale (AIMS) applied by three experienced psychiatrists revealed a score of 16. Under these circumstances risperidone was discontinued and olanzapine 10 mg/d initiated. Two weeks after this treatment approach the AIMS score was established to be just 2, reflecting only slight movements of the tongue, and that the antipsychotic efficacy of the previous agent had been maintained.

Our case possesses two distinct features: tardive dyskinesia induced by the antipsychotic risperidone and emergence of the movement disorder in 9 months' time, quite a short period when classical data is taken into account. Another outstanding aspect of the case is the recovery of tardive dyskinesia after replacement of risperidone by olanzapine.

We must admit that it is unclear whether this recovery was due to discontinuation of risperidone or the pharmacodynamic action of olanzapine, but olanzapine seems to deserve further investigation concerning

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patients suffering from this obscure side effect of antipsychotics. On the other hand, a possible cure of atypical antipsychotic-induced tardive dyskinesia by another agent of the same class may be a cue to debate the 'atypical' concept.

REFERENCES

1 Gwinn KA, Caviness JN. Risperidone-induced tardive dyskinesia and parkinsonism. Mov Disord 1997 ; 12 : 119-21.
2 Lieberman JA. Neuroleptic-induced movement disorders and experience with clozapine in tardive dyskinesia. J Clin Psychiatr Monogr 1990 ; 8 : 3-8.
3 Lieberinan JA, Saltz BL, Johns CA. The effects of clozapine on tardive dyskinesia. Br J Psychiatry 1991 ; 158 : 503-10.
4 Sakkas P, Liappas J, Christodoulou GN. Tardive dyskinesia due to risperidone. Eur Psychiatry 1998 ; 13 : 107-8.