

Tardive Dyskinesia During Treatment with Risperidone

E.-M. Haberfellner

Psychiatrist in Private Practice, Linz, Austria

One of the main problems during long-term treatment with neuroleptic drugs is the development of tardive dyskinesia. The incidence of tardive dyskinesia is about 4–5% per year of neuroleptic treatment. As there is no proven and safe therapy for tardive dyskinesia, the treatments most commonly used are reduction of the neuroleptic dosage or a change to atypical neuroleptics, especially clozapine. Risperidone has been introduced as an atypical neuroleptic drug with low risk for the emergence of extrapyramidal side effects and tardive dyskinesia. Until now few case reports exist about tardive dyskinesia during risperidone treatment (Addington, 1995; Buzan, 1996; Daniel, 1996; Woerner, 1996).

A 23-year-old outpatient was treated for schizophrenia simplex as of November 23, 1995 with 1 mg pimozide. After only a few days the patient discontinued the treatment. Further treatment was started with risperidone on January 11, 1996. The dosage was increased gradually to 4 mg. After a suicide attempt the patient was admitted to a local psychiatric hospital for several weeks. The patient received 20 mg paroxetin and 80 mg prothipendyl for 1 week to induce sleep. Treatment with risperidone was continued. After leaving the hospital the dose was reduced to 3 mg risperidone due to extrapyramidal side effects. On April 22, 1996, the patient reported midly distinct involuntary tongue and jaw movements for the first time. Because the psychiatric condition was stabilized, the dosage was reduced to 2 mg risperidone. In the following weeks the tardive dyskinesia appeared less pronounced but was still detectable. From September to November the patient took his medication irregularly and finally discontinued against medical advice. The patient's last doctor's appointment was on November 11, 1996, when he was hardly impaired by the dyskinesia.

The case presented describes the development of tardive dyskinesia under risperidone treatment. Since the movement disorder began after only three months of continuous neuroleptic treatment, the symptoms are described as "early onset dyskinesia". Contrary to the cases of tardive dyskinesia reported in recent months concerning treatment with risperidone, the cumulative dosage of other neuroleptics administered was in our case very low. It can therefore be assumed that tardive dyskinesia was triggered by the treatment with risperidone.

References

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Dr. Egon-Michael Haberfellner

Friedrichstraße 14
A-4040 Linz
Austria