Risperidone-Induced Tardive Dyskinesia

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This report presents a potential case of risperidone-induced tardive dyskinesia. A 28 year old white schizophrenic male has been under risperidone monotherapy for about one year when he developed dyskinetic movements. There was only a short term exposure to classic antipsychotics prior to risperidone administration.

Until now risperidone has been presented as an "atypical" antipsychotic with few extrapyramidal side effects (*Owens*, 1994; *Marder* and *Meibach*, 1994). Even antidyskinetic effects in doses of 6–16 mg/day have been observed (*Chouinard* et al., 1993). In clinical practice extrapyramidal side effects induced by risperidone can be observed in many cases. So far only a few reports on risperidone-induced tardive dyskinesia have been published. Most of these cases either had long term exposure to classic antipsychotics or had been on additional treatment with risperidone. In contrast to previous reports, the case presented here is of risperidone monotherapy over a period of one year. Previous exposure to classic antipsychotics had been for a short time only.

Mr. A., a 28-year-old white male, was diagnosed with a one-year history of paranoid schizophrenia meeting DSM-IV criteria. He was first hospitalized in November 1995. Prior to admission a neurologist had tried to establish an antipsychotic treatment with zuclopenthixol but the patient did not comply. Hospitalization was necessary due to auditory hallucinations, social withdrawal and recurrent verbal aggressions. Upon admission, results of the physical and neurological examination, computed tomography scan of the head, EEG and routine laboratory screening were normal. The patient was given an antipsychotic regimen of haloperidol 20 mg/day. He remained on this regimen for 16 days, after which the dose was gradually decreased as follows: 10 mg/day for 7 days, 5 mg/day for three days. On the third day of haloperidol treatment the patient suffered a severe dystonic reaction requiring biperiden administration for several days. Mr. A.'s psychotic symptoms completely resolved but he continued to display negative symptoms that even increased. For this reason haloperidol was discontinued after approximately four weeks of treatment. Then a regimen of risperidone was started with an initial dose of 2 mg/day which was titrated over two days to 6 mg/day. Seven weeks after admission the patient was discharged. The risperidone dose was reduced to 5 mg/day in January 1996 and was further reduced to 4 mg/day in June 1996. The negative symptoms persisted and attempts at social rehabilitation were unsuccessful.

In December 1996, the attending physician noted abnormal orofacial movements with chewing and smacking and reduced the risperidone dose to 3 mg/day.

In January 1997, an evaluation of the abnormal movements was performed. The patient was unaware of his moderate chewing and smacking. Choreic movements of shoulders and minimal increase in the muscle tone of the upper extremities was also noted. The score on the Simpson-Angus Scale (SAS) was 8. The Abnormal Involuntary Movement Scale (AIMS) was 12. The Barnes Akathisia Scale (BAS) was 0. The following examinations and analysis were performed to exclude any organic causes of dyskinetic movements and revealed no abnormality: magnetic resonance imaging of the head, EEG, thyroid, renal and hematological functions, serum copper and ceruloplasmin levels, and HIV test. Hepatic function tests showed slight elevation of ALAT and AST. Hepatitis A, B and C antibodies were negative. There was no history of substance abuse.

DSM-IV criteria of tardive dyskinesia were met beyond doubt despite low intensity. The risperidone dose was reduced further, to 2 mg/day. Another physical examination at the end of January 1997 showed some change in the severity and pattern of the abnormal movements. Orofacial dyskinesia was slightly diminished but involved the tongue. Choreic movements of the shoulders were slightly more prominent. Mild abnormal movements of the lower extremities with foot tapping and squirming were also observed. The following values, SAS 4, AIMS 9, BAS 0, were assessed. In contrast to the objective findings the patient reported complete resolution of the movements. His mother assessed him to be more active. The recently established dose of 2 mg/day of risperidone was continued. Another assessment of the patient's dyskinetic movements was made in mid-March 1997, and revealed slight alleviation. It was decided that a change from risperidone to clozapine was not necessary, because of the mild tardive dyskinesia, the patient's personal satisfaction with his condition, and expectation of further improvement.

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Only a few questionable cases of risperidone-induced dyskinesia have previously been reported (*Daniel* et al., 1996; *Buzan*, 1996; *Woerner* et al., 1996). In this case it seems more evident that tardive dyskinesia was induced by risperidone because of the short-term exposure to classic antipsychotics (haloperidol four weeks, zuclopenthixol prescribed in daily doses up to 4 mg but intake mostly rejected). There is still some probability of spontaneous dyskinesia. In view of the pharmacology of risperidone increasing numbers of risperidone-induced tardive dyskinesia should be expected. Further studies are needed in order to assess definitively risperidone's potential for inducing tardive dyskinesia.

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