Tardive dyskinesia induced by risperidone?

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LETTERS TO THE EDITOR

benefit from treatment with anticonvulsants than with "classical" antipanic drugs.

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DONATELLA MARAZZITI, M.D., PH.D. GIOVANNI B. CASSANO, M.D. Pisa, Italy

Tardive Dyskinesia Induced by Risperidone?

TO THE EDITOR: Risperidone, a recently approved antipsychotic drug, is a benzisoxizole derivative that displays potent central antagonism of serotonin (especially 5-HT₂) as well as dopamine (D₃) receptors. Because of its broader spectrum of pharmacologic properties, particularly its 5-HT₂ antagonism, risperidone has been regarded as an atypical antipsychotic that shares with clozapine the potential for superior antipsychotic efficacy and reduced capacity for causing extrapyramidal side effects (1, 2). Although there have been no definitive studies, there is evidence that clozapine has a low propensity to cause (3) and may be effective in suppressing (4) tardive dyskinesia. In one study, risperidone was shown to suppress abnormal movements in patients with tardive dyskinesia (2), but there are no published data on risperidone's tardive dyskinesia-inducing potential. We present a case of a schizophrenic patient in whom the onset of tardive dyskinesia was observed during treatment with risperidone.

Mr. A was a 31-year-old man with undifferentiated schizophrenia who had been treated with trifluoperazine, 10 mg/day, for 1 week before admission to our hospital. Upon hospitalization, he was given a regimen of fluphenazine, 20 mg/day. He remained on this regimen for 10 months, after which the dose level was gradually decreased: 15 mg/day for 4½ months, 10 mg/day for 2 months, 7.5 mg/day for 1 month, 5 mg/day for 2 months, and 2.5 mg/day for 2 months. Benztropine mesylate, 2–4 mg/day, was also prescribed throughout this period.

During this period, Mr. A's psychotic symptoms had completely resolved, but he continued to display negative symptoms and marginal functioning. All medications were discontinued (with his and his family's consent), and he was given no medication for 6 months. He then was started on a regimen of risperidone for treatment of recurring referential and persecutory delusions.

Mr. A's initial risperidone dose was 2 mg/day, which was titrated over 2 days to 6 mg/day. After 4½ months, the dose was increased to 8 mg/day. The next month the dose was increased to 10 mg/day, which was followed by a final increase to 12 mg/day 1 month later; he remained on this regimen for almost 6 months. The risperidone dose then was reduced to 9 mg/day and 1½ months later was further reduced to 6 mg/day, at which it has remained.

Mr. A had been examined every 2 months for the presence of tardive dyskinesia and extrapyramidal side effects. There had been no clinically significant extrapyramidal side effects or abnormal movements during treatment with flu-

phenazine alone or in combination with benztropine, and no movements emerged after medication discontinuation. Before risperidone initiation, while Mr. A was taking no medication, his treating physician had noted finger and ankle movements, but the results of two assessments performed 1 week later by the study raters did not indicate any evidence of tardive dyskinesia. Examination results continued to indicate no presence of tardive dyskinesia until 1 year later, when Mr. A was rated as having questionable tardive dyskinesia. Three weeks later, the presence of tardive dyskinesia was rated as definite (mild severity). Since then, the abnormal movements have persisted, and the latest examination found movements of moderate severity that involved the lips, jaw, tongue, and lower extremities.

A single case can only serve to stimulate interest in further study. However, we believe that this case is unusually clear because of the complete prospective medication data, systematic tardive dyskinesia examinations, and the 6-month drugfree and tardive dyskinesia-free period that preceded the start of risperidone treatment. We are currently enrolling patients who are beginning treatment with risperidone and clozapine to further examine the issue of tardive dyskinesia.

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MARGARET G. WOERNER, PH.D. BRIAN B. SHEITMAN, M.D. JEFFREY A. LIEBERMAN, M.D. JOHN M. KANE, M.D. *Glen Oaks*, N.Y.

Mood Alterations and Tramadol

TO THE EDITOR: Tramadol, a centrally acting analgesic, has recently been introduced into the United States (although it has been available in Germany since 1977). Tramadol has opioid properties, with apparent selectivity at the mu receptor (1). Tramadol also inhibits norepinephrine and serotonin (5-HT) reuptake in rat brain synaptosomes (1). Tramadol is used primarily for acute intermediate-to-severe pain (1) and is believed to have low potential for abuse (2). We report here a case of a patient for whom tramadol treatment seemed to cause alterations in mood.

Ms. A was a 48-year-old white woman with a long history of depression, a suicide attempt 15 years earlier, a history of alcohol abuse (she had been abstinent for the last 6½ years), and Morton's neuralgia (an interdigital neuralgia). Her depression was under control with an oral regimen of fluoxetine, 40 mg/day, and trazodone, 100 mg h.s. Her only additional medication was transdermal patches of es-