Mr. A was a 34-year-old, white male veteran with a history of previous treatment-refractory schizoaffective disorder and amphetamine dependence who was admitted to the hospital for an exacerbation of his depressive and psychotic symptoms. He also reported episodes of irritability, racing thoughts, increasing goal-directed activity, and insomnia, but the differential diagnosis between his amphetamine use and a manic component to his schizoaffective disorder had never been clarified. His only prescribed medication at the time of his admission was quetiapine, 600 mg/day.

At Mr. A's hospital admission, a urine sample was submitted for routine toxicology testing, including analysis for amphetamines, barbiturates, benzodiazepines, cannabinoids, cocaine metabolites, opiates, methadone, alcohol, and tricyclic antidepressants. The results showed a positive toxicology screening for tricyclic antidepressants, whereas the other toxicology assays on the specimen were all negative. Mr. A denied using tricyclics, but given the possibility that surreptitious use of that drug class might be materially contributing to his ongoing clinical symptoms (1), it was important to rule out drug interference in the assay.

The qualitative urine drug tests used in our laboratory are all homogenous enzyme immunoassays (Diagnostic Reagents, Inc.) and are automated on a Hitachi 911 analyzer. The tricyclic assay uses specific antibodies that detect most tricyclic antidepressants in urine, serum, or plasma. A change in the absorbance value that is equal to or higher than that of the calibrator is interpreted as positive. This assay uses a calibrator of 300 ng/ml of nortriptyline, with the calibrator absorbance value arbitrarily set at zero. Mr. A's urine exhibited a tricyclic assay response of 13.

It was noted that quetiapine is similar in structure to the tricyclic antidepressants and so was suspected as a possible interferent. A 25-mg tablet of quetiapine was dissolved in water and diluted to concentrations of 1–10 μg/ml. Levels below 7 μg/ml yielded negative results in the qualitative assay, whereas levels of 7 μg/ml or more yielded positive readings. Thus, the cross-reactivity of this drug in the assay—defined as the concentration of the calibrator divided by the drug concentration that yields a positive result, multiplied by 100—was 4.3% ([300 ng/ml ÷ 7000 ng/ml] × 100).

Another frequently prescribed atypical antipsychotic—olanzapine—was also tested in the tricyclic antidepressant assay, but this drug exhibited minimal cross-reactivity (0.06%–0.07%).

In summary, patients who are prescribed quetiapine can exhibit false-positive test results in assays for urine tricyclic antidepressants. This information will be submitted to the manufacturer of the assay.

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Meige's Syndrome Associated With Risperidone Therapy

TO THE EDITOR: Risperidone is an atypical antipsychotic that is effective for the positive, negative, and cognitive symptoms of schizophrenia (1). The incidence of extrapyramidal signs has been reported to be low during treatment with a dose of 6 mg/day. We report a case of Meige's disease (2–4), an extrapyramidal syndrome that was secondary to risperidone treatment.

Mr. A was a 43-year-old, single Caucasian male who became psychiatrically ill at age 22 years and had since been taking various neuroleptics. From 1980 to 1996, he was prescribed thioridazine, 50 mg/day. More recently, his thioridazine therapy was changed to treatment with risperidone, 6 mg/day, as a result of an exacerbation of his symptoms. Mr. A then started blinking frequently. Sometime later, he noted episodic blepharospasms that either occurred spontaneously or were triggered by stress. As a result, he had to discontinue vision-dependent activities such as driving. More recently, he sought help for blepharospasms.

During an interview, Mr. A was noted to have intermittent blepharospasms that affected his vision. The more he tried to open his eyes, the more tightly his eyelids closed. He often struggled for about 3–5 minutes to open his eyes.

This patient had Meige's disease that was secondary to his risperidone treatment. Because dopamine-2 blocking agents such as neuroleptics have been known to cause Meige's disease (5), this finding is not surprising. In this case, tardive dystonia was ruled out because he had no other evidence of choreoathetotic movements elsewhere in his body. Spontaneous Meige's disease was ruled out because of the absence of a family history of Parkinson's disease, abnormal facial movements, and concomitant physical illnesses such as multiple sclerosis or autoimmune disorders. It is important to recognize this condition because the withdrawal of antipsychotic drugs may lead to a complete recovery from Meige's disease.

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