

RISPERIDONE AND WITHDRAWAL DYSKINESIA

To the Editor:

In light of a recent report on the successful use of risperidone in the treatment of refractory comorbid attention-deficit/hyperactivity disorder (ADHD) and oppositional defiant disorder and conduct disorder, without the presence of an additional psychotic disorder (Kewley, 1999), I would like to add another case report of withdrawal dyskinesia with risperidone.

A.B. is a 13-year-old boy with ADHD, conduct disorder, and an affective disorder with a history of psychotic features (auditory hallucinations, paranoid ideation), who had been treated with lithium, valproic acid, and risperidone. His medical history was noncontributory, he had not been exposed to psychostimulants, and he had no history of substance abuse. Risperidone was begun in December 1998 and titrated to 1.5 mg/day. When his psychotic symptoms had resolved, the risperidone was tapered by 0.5 mg every few months and discontinued in November 1999. Approximately 2 weeks after risperidone was discontinued, the patient developed a withdrawal dyskinesia manifested by mild mouth movements, neck twisting, and intermittent upward gaze.

The patient was begun on low-dose quetiapine, which was titrated to 100 mg b.i.d., and the withdrawal dyskinesia resolved. The quetiapine dose will now be tapered slowly in an effort to prevent a reemergence of dyskinetic movements.

Although withdrawal dyskinesia may resolve within about 6 weeks without any treatment, the concerns raised about the appearance of movement disorders in nonpsychotic children taking risperidone (Demb and Nguyen, 1999) should continue to be evaluated and heeded.

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RISPERIDONE IN ANOREXIA NERVOSA

To the Editor:

The value of medication in the treatment of anorexia nervosa (AN) is uncertain, and to date, no placebo-controlled

studies have demonstrated its utility for weight gain (Jimerson et al., 1996). The delusional fear of weight gain raises the question of whether atypical antipsychotics could be helpful. There is only one case report of risperidone being used in a high-functioning autistic female with AN, but the medication was used to target her paranoia and aggressiveness (Fisman et al., 1996). I describe 2 cases of female adolescents with severe AN who rapidly improved after the addition of risperidone to a selective serotonin reuptake inhibitor.

J.P. is a 19-year-old woman with a 5-year history of AN who denied bingeing, purging, and diuretic or laxative use. At 5 feet 2.5 inches (158 cm), her weight was 80 lb (36.4 kg) and body mass index (BMI) was 14.6 kg/m²; she had secondary amenorrhea and moderate osteopenia. She denied hallucinations or delusions unrelated to her eating disorder. Multimodal treatment including sequential full trials of fluvoxamine, paroxetine, fluoxetine, and sertraline did not help her major depression or weight. In February 1998, at less than 70% of ideal body weight (IBW), she was hospitalized for bradycardia. During a 3-month psychiatric hospitalization, she gained 20 lb but was unable to maintain her weight after discharge; she was hospitalized again 4 months later. Venlafaxine 150 mg b.i.d. lessened her depression, but she continued to lose weight as an outpatient. In December 1998, I added risperidone 1.5 mg/day for her delusional thinking about weight and to avoid a third hospitalization. Within 1 week, J.P.'s anxiety and obsessions about food diminished. During the next month, she gained 16 lb to reach 105 lb, 91% of IBW. Although she was distressed about the rapidity of her weight gain, her depression remitted. Her meal plan was decreased from 3,500 to 2,500 kcal/day, and in April 1999, at 109 lb, 97% of IBW, her menses returned. Over the next 10 months, her focus on body image and weight declined and she remained at IBW with regular menses even after risperidone was tapered and discontinued.

M.R. is a 12-year-old girl with a 2-year history of a restrictive eating disorder with secondary amenorrhea, who denied bingeing, purging, and laxative or diuretic use. She denied hallucinations and delusions unrelated to body image. At 5 feet 1.5 inches (154 cm), 83 lb (37.7 kg), BMI of 15.9, she was 79% of IBW and required medical hospitalization for bradycardia. Sertraline 100 mg minimally helped her anxiety, obsessive thoughts about food, and minor depression. She was unable to gain weight as an outpatient, so to avoid another hospitalization I added risperidone 0.5 mg/day in January 1999. Within 1 week, M.R. could follow her meal plan, was more cheerful and



energetic, and expressed new insight about her anorexia. M.R. increased her risperidone dosage to 0.5 mg t.i.d. as it lowered her anxiety before meals. She gained 8 lb the first month and 4 lb the second month, reaching 95 lb, 91% of 1BW. In May 1999, after her risperidone dosage was decreased to 0.5 mg b.i.d., her insight declined and obsessive thinking about weight returned, but both quickly improved when the risperidone dose was increased to 1.5 mg/day. In September 1999, at 103 lb, her menses returned. She has since maintained her weight and menses for 6 months and reports that she rarely thinks about food or weight.

Both adolescents credited risperidone for their avoidance of another hospitalization and rapid improvement in thinking about food and body image. Both families believed that their child's irritability and abnormal mealtime behavior improved greatly with the addition of this medication. Atypical antipsychotics may be beneficial for patients with AN, even those without clear psychosis, because of the delusional nature of body image distortions and the large weight gain associated with these medications. In addition to increasing appetites, risperidone may slow metabolism as J.P. gained weight despite decreasing her supervised daily caloric intake. Although one would expect that patients with AN would dislike a medication that leads to weight gain, both of these patients wished to continue to take the medication, and M.R. even requested that her dose be raised. They both expressed feeling more "in control" of their thinking while taking the medication.

The only side effect experienced by these 2 adolescents was mild initial sedation. They did not develop dyskinetic movements, and phosphate and liver function test results remained normal. The QTc interval on J.P.'s electrocardiogram (ECG) increased from 400 to 421 milliseconds; thus monitoring ECGs may be indicated in this population. Although risperidone is a medication with potential serious risks, the high morbidity and mortality rates associated with AN may warrant the cautious use of risperidone for some patients.

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NEFAZODONE FOR PTSD

To the Editor:

Few studies have addressed the benefits of pharmacological treatments in children and adolescents with posttraumatic

stress disorder (PTSD). There have been a few articles reporting the possible beneficial effects of clonidine and propranolol in addressing symptoms such as startle response and other arousal symptoms in pediatric patients, but no controlled trials of which we are aware (Famularo et al., 1988; Kinzie and Leung, 1989). Antidepressants—selective serotonin reuptake inhibitors (SSRIs) in particular—have been shown to exert some benefit in adults, but a recent literature review (Donnelly et al., 1999) found no reports of SSRI use in pediatric PTSD. Currently nefazodone is being examined as a possible treatment in adults and has been shown to have some beneficial effects in open trials. We wish to report the potential usefulness of nefazodone in adolescents with PTSD.

Many adolescents with PTSD are brought to the attention of mental health providers because of difficulties with behavior. In fact, many of the patients assessed in our clinics or admitted to our residential treatment programs present not for problems with avoidance, excessive worry, or reexperiencing phenomena, but for disruptive behaviors at home and in the community. We have found that nefazodone may be particularly useful in addressing the symptoms of hyperarousal which bring adolescents to clinical attention. These include improvements not only in anger and aggression, but in restlessness, insomnia, and even concentration. In the context of residential treatment we have observed an improvement in avoidance, anhedonia, and detachment. Our patients often report an improvement in both sleep duration and quality. Improvement in sleep and the symptoms of hyperarousal are consistent with those changes observed in adult patients with PTSD (Hertzberg et al., 1998).

A 1997 case study (Wilens et al.) discussed the efficacy of nefazodone in children and adolescents with mood disorders. The authors concluded that nefazodone was well tolerated and showed some benefit. In our adolescent population we have also found that it is well tolerated in doses of up to 600 mg/day, with an average effective dose of around 200 mg twice per day. While one female patient has reported visual trails at a dosage of 600 mg/day (which resolved with a decrease in dosage), the most common complaints have been of morning somnolence, nausea, and vomiting. These side effects seem to occur more commonly at higher doses. The nausea and vomiting seem to be dose-related and tend to improve with a decrease in dosage. We have, however, had 3 patients (over the course of the past 3 years) who required discontinuation of the medication because of nausea. Somnolence has not been as much of a problem with patients who were significantly activated before its initiation.

Psychotherapy has long been the primary treatment for PTSD, both in adolescents and in adults. It stands to reason that if the problematic symptoms of hyperarousal and avoidance can be attenuated, then psychotherapy may in turn be