High-dose paroxetine treatment for an adolescent with obsessive–compulsive disorder comorbid with Asperger’s disorder

Despite the frequent comorbidity of obsessive–compulsive disorder (OCD) with pervasive developmental disorders (PDD), little evidence is available to guide treatment of such cases. We report a case of OCD comorbid with Asperger’s disorder, in which high-dose paroxetine treatment was effective in improving obsessive–compulsive behaviors. Written parental informed consent was obtained for publication of this report.

A 15-year-old girl with severe contamination fears and contamination-related checking behaviors was admitted to hospital. She was diagnosed as having OCD according to DSM-IV-TR. Several medication trials prior to hospitalization, including fluvoxamine 100 mg/day, risperidone 2 mg/day, haloperidol 6 mg/day, and paroxetine 20 mg/day, had been ineffective in reducing her symptoms. Upon admission the patient’s Yale–Brown Obsessive Compulsive Scale (Y-BOCS) score was 40.

A detailed review of her past history indicated socially inappropriate behaviors since early childhood, such as being uncooperative with other children in preschool. Her obsessive traits became prominent in her grade-school years, spending several hours on simple handwriting assignments to do it as neatly as possible. She was very specific about her dress, sometimes refusing to put anything on because she was unsatisfied with her clothing. Her impairment in social interaction without delay in language and her persistence since childhood led us to diagnose her as having Asperger’s disorder according to DSM-IV-TR, in addition to previously diagnosed OCD. Although obsessive traits had been apparent since childhood, the onset of OCD, which was when contamination fears appeared, was at the age of 15.

Paroxetine was started concurrently with behavioral therapy, and the dose was titrated to 60 mg/day. After 8 weeks her contamination fears began to lessen. During the course of treatment the patient experienced irritability and excess sweating, which diminished after the paroxetine dose was reduced to 40 mg/day in the third month. Her Y-BOCS score at 6 months after initiation of paroxetine was 14. Her obsessive tendencies remained even after her contamination fears almost completely disappeared.

The important role of serotonin in OCD and PDD has been hypothesized from the efficacy of selective serotonin re-uptake inhibitors (SSRI). Results from a randomized, double-blind, placebo-controlled trial indicated that paroxetine is an effective treatment in pediatric OCD, and several other studies have suggested therapeutic benefit of SSRI in PDD. Studies investigating candidate genes related to serotonin regulation, however, such as serotonin transporter gene, have not found any associations in PDD or OCD. Whether obsessive–compulsive symptoms in OCD and PDD have overlapping etiologies remain unclear.

Although concurrent behavioral therapy complicates the evaluation of pharmacotherapeutic effectiveness, it is inferred that paroxetine showed dose-dependent efficacy for OCD in a patient with Asperger’s disorder. Considering the evidence of efficacy in both disorders, pharmacotherapy with SSRI is recommended in such patients.
There are no guidelines, however, for treatment of OCD symptoms in those diagnosed with PDD, and thus patients for whom the pharmacotherapy could be beneficial may not be receiving adequate treatment. We believe that clinical trials are warranted to clarify the benefits versus the risks of using SSRI in children and adolescents dually diagnosed with PDD and OCD.

It is noteworthy that, in the present case, the contamination fears disappeared with paroxetine treatment while obsessive traits of PDD, which had been present since before the onset of OCD, remained. Further studies may clarify the difference between obsessive symptoms observed in PDD and OCD in regard to medication response, which may lead to the drawing of a distinction between the two disorders.

REFERENCES


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