CITALOPRAM AND MANIA

To the Editor:

Treatment-emergent mania has been reported with various selective serotonin reuptake inhibitors (SSRIs) (Diler and Avci, 1999; Grubbs, 1997). We report four cases of citalopram-induced mania in children.

Case 1. A 6-year-old boy with a family history of obsessive-compulsive disorder (OCD) was diagnosed with severe depression without psychotic symptoms. He developed mania without psychotic symptoms after taking citalopram 20 mg/day for 7 days. Although he immediately improved on stopping citalopram, complete remission occurred only with lithium 600 mg/day (levels 0.8–1.1 mmol/L).

Case 2. A 15-year-old boy with a family history of recurrent depressive disorder had moderate depression and a suicide attempt. His previous treatments included fluoxetine 40 mg/day and imipramine 25 mg/day. Because he reported obsessions and compulsions, these were stopped and citalopram was titrated to 30 mg/day. Over the next 2 weeks, all his symptoms remitted completely. Four weeks after starting citalopram, the child developed mania without psychotic symptoms. When he presented to us 3 weeks later, we stopped citalopram. Although he immediately improved, complete remission occurred only with lithium 600 mg/day.

Case 3. A 14-year-old girl with no family history of psychiatric disorder presented with 3-year history of untreated OCD. We titrated fluoxetine to 50 mg/day over 3 months. Two weeks later, she developed mania. We stopped fluoxetine, but complete remission occurred when risperidone 2 mg/day and lithium 600 mg/day (blood levels 0.7 mmol/L) were added. However, her OCD reappeared. A retrial of fluoxetine 20 mg/day was attempted. The child's manic symptoms reappeared after 6 days. Thereafter, fluoxetine was stopped. Lithium and risperidone were titrated to 900 mg/day and 3 mg/day, respectively. Although the manic symptoms remitted completely, her OCD reemerged with greater severity. Citalopram 10 mg/day was added with dramatic improvement in her OCD. The child developed hypomania after a week, and citalopram was stopped. As OCD reemerged, the child's general practitioner restarted citalopram at 10 mg/day, and hypomania continued. Over the next 1 week, the child reached euthymia without any additional interventions. We continued her on citalopram 10 mg/day, lithium 900 mg/day, and risperidone 3 mg/day with no residual symptoms.

Case 4. A 15-year-old boy with a family history of depression and two suicides had severe depression, catatonia, and social phobia. His previous treatments included fluvoxamine 150 mg/day, olanzapine 2.5 mg/day, and fluoxetine 20 mg/day. Then his general practitioner stopped the SSRIs and started a combination of citalopram 20 mg/day, escitalopram 10 mg/day, and olanzapine 2.5 mg/day. The child developed mania 13 days later that lasted 1 week. We stopped citalopram, escitalopram, and olanzapine, after which depressive symptoms with catatonia reappeared. The child was treated with sertraline 225 mg/day, electroconvulsive therapy, and behavior therapy for social phobia. After 2 months, depression and catatonia remitted, but social anxiety continued.

The manic symptoms by which *DSM-IV* diagnoses could be made were irritable or euphoric mood, pressure of speech, increased activity, high-energy levels, aggression, recklessness, overfamiliarity, increased socialization, grandiose ideas, and decreased need for food and sleep. In all the children, the manic symptoms temporally correlated with the use of citalopram. All improved to an extent on stopping citalopram. However, all required further treatment.

The patient in case 3 demonstrated manic symptoms with more than one SSRI. This was dose related. This has not been well documented. This case also raises the possibility that a treatment-emergent mania with one SSRI may sensitize a patient to treatment-emergent mania with lower doses of another SSRI. As demonstrated by case 3, citalopram could be used despite causing hypomania if such symptoms are controlled.

The patient in case 4 was on both citalopram and escitalopram when mania emerged, raising the possibility of escitalopram also being a reason for the same.

We corroborate the observation of Heimann and March (1996) that nonremission of the mania to stopping of SSRIs in cases 1, 2, and 3 suggests the possibility of the unmasking of a latent bipolar illness rather than a drug-induced switch. Case 4, however, suggests a citalopram-induced switch.

No child had a family history of bipolar disorder. Two had a family history of depressive disorders, raising the possibility of a predisposition to treatment-emergent mania. The patient in case 3 had hypomania with two SSRIs but no family history of a psychiatric disorder. We advocate that caution be exercised in treating depression with citalopram and escitalopram in pediatric populations even in the absence of a family history of affective illness.

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