Dear Editor,

This report suggests that emergent behavioral disinhibition during antidepressant treatment may represent milder expression of hypomania.1

The patient was a 33-year-old, married, Caucasian man with a 2-year history of chronic depression. He was an executive and became disabled due to depression. Laboratory work-up, including complete blood count, creatine kinase level, electrolytes, renal, liver and thyroid function tests, was unremarkable.

Since this patient showed partial response to previous treatment with paroxetine (40 mg/day), a Selective Serotonin Reuptake Inhibitor (SSRI), a second trial with another SSRI was considered. Citalopram treatment was initiated at the dose of 20 mg/day and gradually titrated to 60 mg/day over a period of twelve weeks. The patient responded partially to high doses of citalopram. He appeared normothymic on subsequent monthly follow-ups for 3 months, but remained with social isolation, fatigue, loss of interest and pleasure.

His wife found that he had been shopping a lot. He seldom stayed at home during the recent weeks. He displayed irritable reactions when his behavior and whereabouts were questioned. She also found that he had spent 1,850 dollars in clothes and shoes. This behavior of excessive spending was considered to be totally out of his character. There was no previous or family history of bipolar personality, substance abuse, and other impulse-control disorders. Interestingly, the psychiatric evaluation performed during the period of problematic behavior did not show any evidence suggestive of persistent hypomanic mood (irritability, elation lasting for at least 1 week). In addition, cross-sectional mental status examination did not reveal pressure of speech, flight of ideas or grandiosity.

Subsequently, citalopram was decreased from 60 to 20 mg in a week. The energized risk-taking and excessive spending behaviors disappeared in 2-3 weeks after the reduction of citalopram. However, this patient did not meet the DSM IV criteria for hypomania or mania due to the absence of historical or clinical evidence of persistent hypomanic moods (elevated, irritable).

Discussion

Antidepressant-associated mania was found to be milder in psychopathologies with less severe levels of psychotic symptoms, psychomotor agitation and bizarre behavior than spontaneous mania. However, there were no differences in phenomenological and nosological status between antidepressant-induced hypomania and spontaneous hypomania in bipolar II.2,3

Consistent with the above case report, antidepressant-associated hypomania may also be predominately manifested with behavioral changes rather than with hypomanic moods. This awaits further investigation. Interestingly our patient exhibited mainly disinhibition symptoms, including irritability, impulsivity and expensive pending, but not mood related symptoms. It is interesting to remark that some authors have proposed two different factors in hypomania, an active/elevated factor where mood elation is a core symptom and the risk-taking/irritable factor whose symptoms are very close to those presented by our patient.4,5

Careful clinical enquiry about the behavioral indicators of hypomania during treatment with antidepressants may facilitate earlier identification and appropriate management of antidepressant-associated hypomania. The major concerns in recognizing the existence of minor forms of antidepressant-associated hypomania will be overdiagnosis of hypomania secondary to antidepressant treatment or bipolar disorder with the resulting increased utilization of mood stabilizers. An adjusted clinical assessment is necessary to minimize this overdiagnosis of antidepressant induced secondary hypomania on the basis of hypomanic behavior as discussed previously. However, the recognition of the nosological status of antidepressant-associated hypomania as bipolar type III may contribute to overdiagnosis of bipolar disorder.4,6

References