Hypomania as a Genuine Side Effect of Fluoxetine

To the Editor: Antidepressant-induced manic switch is generally seen in patients with risk factors for bipolar disorder. To the contrary, the authors hereby report a case of an adolescent boy who developed fluoxetine-induced hypomania but did not have any risk factor for bipolarity. Hypomania might be a genuine side effect associated with antidepressants like fluoxetine.

Literature suggests that antidepressants can precipitate mania in patients with unipolar/bipolar disorder.1 During the pre-drug era, spontaneous mild depression after a manic episode and spontaneous hypomania after a melancholic episode were common, and had no bearing on the primary diagnosis.2 Previous reports of fluoxetine-induced mania/hypomania have been mostly in patients with depression.3,4 It can be argued that these cases had natural manic episodes of bipolar disorder. On the contrary, fluoxetine has been argued to be useful in bipolar II disorder.5 We hereby report a case of an adolescent boy who developed hypomania while on fluoxetine without any risk factor for manic switch.

Case Report
A 17-year-old boy was suffering from tension-type headache along with mild episodic anxiety symptoms. His past, family, and personal history were nonsignificant. There was no history to suggest depressive features. He was started on fluoxetine 20 mg per day along with relaxation exercises. The patient followed up after a period of 3 weeks with complaints of decreased need for sleep, talkativeness, over-familiarity, increased activity levels, and demanding behavior for the past 4 days. On examination, he had increased psychomotor activity, euphoric affect, racing thoughts, grandiose ideas, and inflated self-esteem. No other significant history was elicited. All his biochemical parameters, including CT head scan, were normal. Diagnosis of fluoxetine-induced hypomania was made. Fluoxetine was stopped, and clonazepam 1 mg was started. The patient improved in hypomanic symptoms in a week’s time. He was then followed up, and he continued reporting headache. Amitriptyline was started, gradually increased to 25 mg per day. The patient has been maintained on this dose for the last 8 months without any recurrence of hypomania.

Discussion
Previous reports of hypomania/mania on fluoxetine have been in cases with a diagnosis of depressive disorder or those with risk factors for bipolar disorder.3,4,6,7 Ours was a case of tension-type headache with nonspecific anxiety symptoms, and did not have any features of depression or any risk factors for bipolar disorder.

We could find only few case reports of mania being induced by SSRIs in patients with obsessive-compulsive disorder (OCD) with or without mood disorder.8,9 OCD is also commonly associated with depressive disorder, and recent literature has pointed toward comorbidity of OCD with bipolar disorder.10,11 Probability of mania or hypomania later was remote in this case because of the absence of various risk factors for bipolar disorder.12,13

The absence of depressive disorder or family history of bipolar disorder, temporal correlation between fluoxetine therapy and onset of hypomania, and quick recovery with a low dose of benzodiazepine after complete withdrawal of fluoxetine shows that this is a case of true fluoxetine-induced hypomania.

Unique points of our case are the facts that the patient did not have any depressive disorder or risk factor for mania and the fact that the patient had been well-maintained on amitriptyline. Previous reports have suggested that tricyclic antidepressants like amitriptyline are more commonly associated with manic switch than are SSRIs.14 The biological mechanism of SSRI-induced mania remains unclear, although serotonergic and catecholamine mechanisms have been implicated.15

Tension-type headache is not even a probable risk factor for antidepressant-induced mania or hypomania. It can be argued confidently that previous case reports may have been having natural bipolar disorder and in other cases, probability of mania or hypomania in future cannot be ruled out.

Our case thus represents genuine fluoxetine-induced hypomania. More such reports would help us to differentiate the drug-induced mania or hypomania from a natural switch.

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