

LETTER TO THE EDITOR

A Case of Hypomania Induced by Lamotrigine Augmentation

To the Editor: The induction of mania or hypomania has been infrequently reported for anticonvulsants, but has been described for divalproex,¹ zonisamide,² topiramate,^{3,4} gabapentin,^{5,6} and lamotrigine—most often in the context of the treatment of bipolar depression.^{7–10} To our knowledge, lamotrigine-induced hypomania has been reported in unipolar depression on only one occasion, when used as an augmentation strategy with bupropion for depression in a 23-year-old woman.¹¹ In that single case report, the patient initially experienced an improved mood on lower doses of lamotrigine (ie, 25 mg and 50 mg) but, 1 week after an increase to 75 mg/d, developed hypomania. When the dosage of lamotrigine was decreased to 50 mg/d, the hypomanic symptoms subsided.

In the following case report, we describe a second case of hypomania that developed when lamotrigine was prescribed as an augmentation strategy in a patient with unipolar depression.

Case report. Ms A, a 31-year-old woman, initially presented in 2008 for the treatment of major depressive disorder (per *DSM-IV* criteria) characterized by dysphoric mood, anhedonia, decreased energy/fatigue, impaired concentration, hypersomnia, psychomotor slowing, low self-esteem, feelings of hopelessness, and general dissatisfaction with life. At evaluation, the patient denied any manic episodes, appetite/weight changes, suicidal ideation, drug/alcohol abuse, or psychotic symptoms. The family history indicated depression as well as antidepressant treatment in mother and father. Several depressive symptoms had been present for longer than 2 years, confirming the presence of *DSM-IV* dysthymic disorder, and the patient evidenced some obsessive-compulsive personality features as well.

Ms A had been on treatment with a number of antidepressants in the past, but at the time of lamotrigine exposure had been on duloxetine 60 mg/d for 1 year. (She had also received several past augmentation strategies including bupropion, buspirone, gabapentin, and topiramate.) Because she was experiencing some mood slippage, lamotrigine was initiated in June 2010 as an augmentation strategy. Lamotrigine was started at 12.5 mg/d and then increased by 12.5 mg every 2 weeks to 50 mg/d. Initially, the patient experienced an improvement in mood. However, 2 weeks after she reached a dose of 50 mg/d, the patient reported feeling “hyper.” Ms A could not sleep but did not feel a need for sleep, was easily distracted (eg, would jump from task to task without completion), could not engage in sedentary activities such as watching television, started talking faster (a client teased her about excessive coffee ingestion), experienced excessive psychomotor activation (eg, she started working out twice daily), became inappropriately flirtatious on one occasion, and ran through a fountain and sustained a minor injury. A diagnosis of hypomania was made and, as in the preceding case report, the lamotrigine dose was decreased (to 37.5 mg/d in this case), leading to the recession of symptoms.

The induction of hypomania by lamotrigine in a patient with unipolar depression prompts clinicians to be alert to such paradoxical phenomena when prescribing anticonvulsants. Ironically, the anticonvulsants, many of which have formal indications by the US Food and Drug Administration for the treatment of mania/hypomania, can on rare occasion induce these unexpected symptoms.

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