Psychological Medicine (2013), **43**, 895.

Correspondence

Psychological Medicine, **43** (2013).
doi:10.1017/S0033291712003005
First published online 10 January 2013

Letter to the Editor

Shopping frenzy induced by naltrexone – a paradoxical effect in bipolar disorder?

The mu-opioid receptor antagonist naltrexone is used to treat alcoholism, depersonalization disorders and impulse control disorders. Positive effects of naltrexone on compulsive buying have been reported (Grant, 2003). We report a paradoxical effect with the induction of manic symptoms and excessive buying activities under naltrexone.

Mr A, a 48-year-old man with recurrent major depression (MD), was hospitalized in September 2011 for depressed mood, agitation, inflated self-esteem, grandiosity, flight of ideas, distractibility and unrestrained buying [Beck Depression Inventory (BDI) 21 points; Hypomania Checklist (HCL-32-RI) 20 points]. We diagnosed mixed state bipolar disorder with both depressive and manic symptoms [International Classification of Diseases (ICD)−10: F31.6; Diagnostic and Statistical Manual of Mental Disorders (DSM)-IV: 296.63]. Venlafaxine (150 mg/day) for recurrent MD commenced in 2008. Naltrexone (150 mg/day) for depersonalization commenced in 2008, as co-morbid post-traumatic stress disorder was then suspected. Retrospectively, we could not corroborate this diagnosis, apart from dissociative symptoms and insomnia. Between 1981 and 2008, approximately 12 depressed and three hypomanic episodes were reported by the patient. After starting naltrexone in 2008, both the severity and frequency of (hypo-)manic episodes increased to two episodes/year, i.e. eight episodes until admission, while depersonalization ceased. During the current hospitalization, we substituted venlafaxine for citalopram, initiated valproate up to 2000 mg/day, and added olanzapine up to 20 mg, reducing mood fluctuations and thought disorder (BDI 14 points; HCL-32-RI 16 points). However, excessive buying remained unaffected. Cognitive–behavioural techniques to reduce excessive buying were not successful, because of ongoing distractibility. Due to the striking increase in manic episodes after naltrexone initiation, this was withdrawn without reoccurrence of depersonalization. However, we observed a significant reduction in excessive buying and mood stabilization about 2 weeks later. Mr A was increasingly able to implement behavioural changes in daily life. Due to weight gain, we substituted olanzapine for aripiprazole (15 mg/day), with stable treatment effects (BDI 11 points; HCL-32-RI 11 points). Mr A was discharged and 4 weeks later, his mood remained stable and compulsive buying had terminated.

Previously reported side-effects of naltrexone in bipolar disorder with alcohol dependency include such opioid-withdrawal symptoms as nausea, dizziness, dysphoria (Sonne & Brady, 2000), and hypomanic symptoms in cannabis users after heroin detoxification (Sullivan & Nunes, 2005). Our patient reported no history of substance abuse, but manic symptoms developed with the commencement of naltrexone. Naltrexone acts by blocking mu-receptors expressed in brain regions involved in stress response and emotional regulation. Our report suggests that mu-receptor blockade plays a pathogenetic role in mania, even if the opiate system is unaltered by substance abuse. Contrary to reported positive effects of naltrexone on compulsive buying (Grant, 2003), our case study reveals that the converse effect may occur in bipolar disorder. Due to the potential to induce manic symptoms, caution should be exercised in using opioid antagonists in bipolar disorder.

Declaration of Interest

None.

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