CASE REPORT

Antidepressant Triggering Mania in Bipolar Disorder

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ABSTRACT

Mood elevation by antidepressants is considered by DSM IV(Diagnostic and Statistical Manual of Mental Disorders) as a bipolar disorder, whereas earlier it was considered as a drug induced reaction. Risk of mood switching with anti depressant treatment is observed more in adolescence and early 20s rather than adults probably because adults with bipolar disorder are usually aware of their diagnosis. Elevation of mood is seen to be more common with TCAs (Tricyclic Antidepressant) than SSRIs (Selective Serotonin Reuptake Inhibitors). Bipolar individuals, taking an antidepressant, should also be on a mood stabilizer or atypical antipsychotic along with antidepressant. The case reported here is that of a young man aged 20, presenting clinical features of social withdrawal, lack of concentration, fear of the unknown in facing people and hesitancy in attending large gatherings. Being diagnosed as social phobia when started with anti depressants, within a month's time ended in severe manic phase with blooming psychosis.

KEY WORDS: *Cognitive Behavior Therapy(CBT), Tricyclic Antidepressant (TCA), Bipolar Disorder (BPD), Selective Serotonin Reuptake Inhibitors (SSRIs).*

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INTRODUCTION

Treatment emergent mania is very destructive for the patients and their families. Treating mixed episodes of bipolar disorder is a challenge for the physicians as it can heighten the risk of mood cycling. 1 in 100 is diagnosed with this condition. Bipolar disorder can occur at any age, although it often develops between the ages 18 and 24 years. Men and women from all backgrounds both are likely to develop bipolar disorder. The pattern of mood swings in bipolar disorder varies widely between people with some being stable most of the time and having only a couple of bipolar episodes in their lifetime, while others may experience many episodes.[1]

CASE

A 20 year old unmarried man reported to the family physician with complaints of social withdrawal and fear of the unknown while speaking public. These symptoms increased gradually to severity over the course of a year. Ultimately the patient stopped attending the college. The inappropriate behavior was noted by the family members. The patient also underwent significant weight loss over the past six months due to lack of appetite.

The patient had indulged in smoking of cigarettes and substance abuse of charas six months ago, which he quit later on. Patient history revealed a normal birth with subsequent good academic performance in school graduating with excellent grades with no complaints from his teachers or peers. However after the commencement of college he developed symptoms of social withdrawal and lack of concentration. The past history revealed the grandfather had some psychiatric illness but due to insufficiency of data was insignificant.

The patient, on appearance, was a presentable man having competent speech. He denied being depressed while but affirmed inability to concentrate and perform routine activities. He suffered from guilt of being engaging in substance abuse with concerns over personal religiosity. The patient did not suffer from delusions, hallucinations or compulsions and depicted lack of suicide ideation.

A provisional diagnosis of social phobia was made, consequent to which education and counseling was provided. Non-pharmacological strategies of preparing speeches were elaborated upon, including rehearsing in front of mirror and presenting to small group of people graduating to a bigger audience. Breathing and relaxation exercises were taught in addition to counseling for improving self confidence. The patient was referred to a clinical psychologist for psychotherapy and CBT (Cognitive Behavioral Therapy) for improvement of his symptoms.

Post review from a tertiary care psychiatrist, the psychologist commenced the psychotherapy sessions along with TCA, Clomipramine starting with 25mg at bedtime for the first week and increasing to 50mg, 75mg and 100mg by week 2, 3 and 4 respectively. The patient was compliant with medications and psychotherapy sessions but no improvements were noticed. After 3 weeks of medical treatment, the patient showed abnormal behavior with abrupt ideas of goal accomplishment. His speech became pressurized and fast and seemed to jump from one idea to another accompanied with decrease need for sleep. There were no delusions and hallucinations but the patient grew more religious supporting a beard and wearing a cap. He recited quranic verses loudly all day long and within a period 2-3 days these symptoms aggravated. The psychiatrist stopped the antidepressant, and diagnosed the condition as hypomania. He was prescribed quitepine 25mg thrice a day, lorazepam 0.5 mg thrice a day and topiramte 25 mg twice a day. The sedatives had little effect with the patient not being able to sleep and ultimately ending up in mania with psychosis supported by abusive and aggressive, uncontrollable behavior. The patient was again rushed to the psychiatrist, and admitted for 2 weeks with treatment of injectable sedatives and with his hands tied up. He was put on Olanzepine 10 mg twice a day , a mood-stabilizer Sodium Valproate 1,000 mg twice a day and lorazepam 1 mg at bed time. His condition improved but akathesias occurred leading to severely a disturbed behavior which was managed by propanolol and anticholinergics. Gradually with occupational and vocational training and a lot of family support, the patient improved. Now after six months of regular psychiatric family physician's treatments he has rejoined his college and is able to carry out all his responsibilities smartly.

The patient is compliant with his medications and leads a happy life.

DISCUSSION

Medications such as antidepressants can trigger a manic episode in people susceptible to bipolar disorder requiring careful treatment. As depressive episode can turn into a manic episode when an antidepressant medication is taken, an antimanic drug is recommended to prevent a manic episode. The antimanic drug creates a 'ceiling', partially protecting the person from antidepressant-induced mania.[2]

A study in 2002 showed antidepressant-induced mania or hypomania wast in 39.6% (21/53) of the study group. Patients who developed manic features soon after starting an antidepressant had more antidepressant trials per year than those who did not ($p < 0.05$). A history of substance abuse and/or dependence was associated with substantially increased risk for antidepressant-induced mania (odds ratio = 6.99, 95% CI = 1.57 to 32.28, $p = 0.007$).[3]

When a person has been diagnosed as having depression, he or she may be prescribed anti-depressants that may take up to three weeks to work. A person may then be pushed into a state of mania, also referred to as medication induced bipolar disorder, especially if larger doses of anti-depressant were required. Anti-depressants may cause a manic episode in those who are vulnerable to bipolar disorder. In those cases, the patient is prescribed a mood stabilizer along with the anti-depressants to prevent further manic episodes.[4]

The occurrence of mania during anti depressant treatment is a key issue in the clinical management of Bipolar disorder.[5] A person must be diagnosed as having unipolar depression or bipolar depression before embarking on a treatment plan.

Quite a few depressed patients actually worsen on antidepressant drugs, become agitated, irritable, and angry, yet clinicians do not recognize that change as a switch to irritable mania or hypomania, or a mixed depressed state. Patients suffering mania or hypomania are expected to be euphoric and expansive, However, the fact that almost one-half of bipolar mania presents with irritability is not widely recognized.[6]

Some patients with depression manifest anger and hostility. It is not widely recognized that treating bipolar depression with an antidepressant might lead to any of four undesirable switches: mania, hypomania, mixed state, or rapid cycling. Patients with all of these complications are simply labeled for their condition as “treatment-resistant depression,” especially if the patient switched to rapid cycling with recurrent depressions (which often happens with BD II patients who receive antidepressant monotherapy).[6]

Relative to the general population, individuals with severe psychotic disorders have increased risks for smoking (odds ratio, 4.6; 95% CI, 4.3-4.9), heavy alcohol use (odds ratio, 4.0; 95% CI, 3.6-4.4), heavy marijuana use (odds ratio, 3.5; 95% CI, 3.2-3.7), and recreational drug use (odds ratio, 4.6; 95% CI, 4.3-5.0). All races/ethnicities (African American, Asian, European American, and Hispanic) and both sexes have greatly elevated risks for smoking and alcohol, marijuana, and drug use.[7]

A study in 2001 showed switches to hypomania or mania occurred in 27% of all patients (N = 12), (24% of the subgroup of patients treated with SSRIs [8/33]); 16% (N = 7) experienced manic episodes, and 11% (N = 5) experienced hypomanic episodes. Sex, age, diagnosis (bipolar I vs. bipolar II), and additional treatment did not affect the risk of switching.[8]

Evaluation of a bipolar disorder should address appearance, affect/mood, thought content, perceptions, suicide/self-destruction, homicide/ violence/aggression, judgment/insight, cognition, physical health, complications (example suicide, homicide, and addictions)[9].

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