CASE REPORT

Antidepressant Induced Mania: Is it a risk factor for Antidepressant abuse?

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ABSTRACT

Induction of mania is a common occurrence with antidepressant use. A case of antidepressant induced hypomania leading to antidepressant abuse is presented. The clinical implications of antidepressant abuse in bipolar disorder are discussed.

Key words: Antidepressant abuse, hypomania, bipolar disorder.

INTRODUCTION

Induction of mania with antidepressant drug treatment is a common and complex problem. The occurrence of this phenomenon has been reported not only in patients with bipolar or unipolar disorder but also in obsessive-compulsive disorder (OCD) (Perugi et al; 1997, Swartz et al; 1999).

In patients of OCD with comorbid bipolar disorder, the prescription of antiobessional drugs alone can lead to mood destabilization and its consequent complications such as non-compliance on mood stabilizers and even antidepressant abuse. We are reporting here a case of OCD with bipolar disorder with coexistent antidepressant abuse.

CASE REPORT

A 28 year old engineer with no past history or family history of psychiatric illness presented with a 12 year old illness, which began with obsessive fears of causing physical harm to his father. These thoughts caused much distress to the patient. For these symptoms the patient sought a psychiatric consultation after about 1-month of illness. He was provisionally diagnosed as a case of OCD and was started on T. clomipramine (150 mg/day). Within 2 weeks of starting clomipramine the patient underwent a hypomanic switch characterized by increased confidence, increased self-esteem, elated mood, decreased need for sleep and increased energy with a significant decrease in his obsessive symptoms. When the clomipramine was tapered, the patient started complaining of low mood, lassitude, increased fatigue and worsening of his obsessions. He also reported missing the "high" induced by clomipramine.

The obsessive symptoms changed in nature over the last 10 years. Currently they are elaborate checking and counting rituals. For these obsessive symptoms the patient had received several trials of antiobessional drugs including trials with fluoxetine (20-40 mg/day), sertraline (50-150 mg/day), fluvoxamine (100-200 mg/day) either individually or in combination for several weeks. In addition he had also received trials of amineptine (100-200 mg/day) and venlafaxine (12.5-150 mg/day).

During these trials, the patient had experienced hypomanic symptoms with amineptine, fluvoxamine and venlafaxine. These hypomanic symptoms would start within 3-4 days of treatment initiation and could last for about 1-2 weeks (maximum 1 month). In this period the patient describes himself as at his creative best with marked increase in his work productivity, increased libido and a top of the world feel ("I am so confident that I feel I can walk into a cabinet meeting") with significant reduction in his obsessive symptoms. Even though the treating psychiatrist stopped the antidepressant drug on all occasions, the patient continued to surreptitously use the same to experience a high. The misuse of the antidepressant was almost always done at crucial times of his life when he felt that the extra energy and confidence were more than useful. He experimented with a variety of antidepressants and reported that for him, amineptine would reliably ("9 times out of 10") induce a high.

When amineptine was withdrawn from the Indian market he started misusing fluvoxamine and venlafaxine. The latter two drugs would not however reliably induce a high.

Six years ago the patient was started on Lithium (600 mg/day; serum levels 0.78 meq/lit) with a provisional diagnosis of bipolar disorder NOS (DSM-IV; 1994). The patient is however non-compliant on lithium. Currently sodium valproate (1000 mg/day; serum levels 70 mg/ml) has been co-prescribed with lithium on which again compliance is doubtful.

DISCUSSION

There is a relatively sparse body of literature regarding antidepressant abuse (Haddad; 1999) Most of this literature is regarding amineptine abuse (Biondi et al; 1990). The susceptibility to develop a hypomanic switch with antidepressants may be either due to the pharmacological property of the drug, individual susceptibility or both. Amineptine is a selective dopamine reuptake inhibitor (Barattini; 1997) The amphetamine like effect of this drug may be responsible for its abuse liability. Antidepressant induced mania tends to be milder and more time limited than in patients with spontaneous mania (Stoll et al; 1994). Antidepressant induced hypomania may be dependant on the dose of the antidepressant drug (Ramasubbu R; 2001) and a premorbid hyperthymic temperament (Henry et al; 2001). Bipolar patients with the "S" variant of the 5HTTLPR
Polymorphism of the serotonin transporter protein gene may be at a higher risk of antidepressant-induced mania (Mundo et al.; 2001). Such patients may also be non-compliant on mood stabilizers. Clearly there is a subgroup of patients with an underlying bipolar diathesis who may misuse antidepressants. Till such patients are characterized both clinically and biologically, it is imperative to enquire for spontaneous or drug induced hypomania before starting a patient on antidepressant drugs.

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