FLUOXETINE INDUCED HYPOMANIA- A CASE REPORT

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Recently, many cases of Fluoxetine induced mania have been reported in the literature. The detailed analysis of these case reports demonstrates that these patients had either positive family history of affective disorder (Lebegne, 1987), past history of affective illness (Settle and Settle, 1984; Lebegne, 1987; Nakra et al., 1989) or were related to high doses of fluoxetine (Chouinard and Steiner, 1986; Turner et al., 1988). Fluoxetine might have precipitated mania in susceptible individuals (genetic loading) or mania might have developed naturally at that time even without fluoxetine (patients with past history of affective disorder) in these patients.

This case report is different from the earlier case reports.

Mr. M.L., 41 year old male graduate, was brought with 20 years history of repeated hand washing, checking and other obsessive rituals interfering with his personal, family and occupational areas and patient was totally incapacitated. This patient fulfilled DSM-III-R criteria for obsessive compulsive disorder. Patient did not report significant depression at any time during the course of illness. Family history was negative for any psychiatric disorder including alcohol and drug abuse.

Before coming to this hospital, the patient received adequate treatment with clomipramine, amitriptyline and imipramine without much change in symptomatology. Most of the antidepressants were associated with anticholinergic side effects. Initially, the patient was started on behaviour therapy (Flooding with response prevention and thought stopping) Behaviour therapy was continued for about 3 months (28 sessions) without much benefit.

At this stage, Fluoxetine was added to behaviour therapy and the dose increased to 60 mg per day in 3 weeks. After 4 weeks of starting Fluoxetine, the patient reported improvement in obsessive rituals and obsessive thoughts. Improvement was also evident to other family members.

One week later (after 5 weeks use of Fluoxetine), patient demonstrated unusual cheerfulness, increased socialization, decreased desire for sleep, overconfidence and felt overenergetic. The patient discontinued Fluoxetine on his own. The above symptoms lasted for about 10 days even after discontinuation. A diagnosis of hypomania induced by Fluoxetine was made.

After two weeks, Fluoxetine was restarted and gradually increased to 60 mg per day. This time the patient did not demonstrate any hypomanic/manic symptoms. Fluoxetine was further increased to 60 mg per day and one week later patient demonstrated overspending, irritability, excessive intake of food, decreased sleep, overtalkativeness and agitation. The patient met DSM-III criteria for hypomanic episode. Fluoxetine was stopped and hypomanic symptoms subsided within 14 days. Currently the patient is on 40 mg of Fluoxetine per day without hypomanic symptoms and continues to report sustained improvement.

DISCUSSION

This case report highlights the following:

Fluoxetine is well accepted as compared to conventional tricyclic antidepressants and this is compatible with an earlier study (Cooper, 1988). Fluoxetine is effective in patients with obsessive compulsive disorder. (Turner et al., 1988; Fontaine and Chouinard, 1986; Jenike et al., 1989 and Pigott et al., 1990) and thus indirectly supports the serotonergic hypothesis of obsessive compulsive disorders. Fluoxetine might induce mania/hypomania even in non-susceptible individuals. In contrast to earlier case reports of Fluoxetine induced mania and hypomania, our patient did not have any family history of affective disorders and had never experienced depressive or mania episode in the past. Occasionally, Fluoxetine is reported to produce suicidal ideation and there are two cases of successful suicide with Fluoxetine overdose reported in the published literature (Teidia et al., 1990).

Further research and clinical use of Fluoxetine might clarify the issue. The mechanism by which
fluoxetine produces these two diametrically opposite reactions is obscure.

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