Akathisia is a common side-effect of the neuroleptic drugs and occurs in about 5% to 75% cases treated with these compounds (Barnes et al., 1985; Van Putten et al., 1984). In addition, akathisia like syndromes are produced with conditions such as uremia, idiopathic restless legs syndrome, congestive heart failure, idiopathic parkinsonism, and with drugs such as reserpine, tricyclic antidepressants (Gibb et al., 1986; Zubenko et al., 1987) and diltiazem.

In 1978, Zitrin et al. reported that 18% of their cases in a series of patients with phobias, treated with imipramine developed a syndrome characterised by "insomnia, jitteriness irritability and unusual energy" on dosage ranging from 5-75mg/day. This is quite similar to akathisia, a side-effect also reported with the antidepressant fluoxetine by Lipinski et al. (1989).

A single case report of akathisia as a side-effect with fluoxetine is being discussed below.

CASE HISTORY

A 22 year old unmarried, north Indian male, visited our hospital clinic, with 3 year illness history, diagnosed as obsessive compulsive disorder (OCD) with secondary depression. He was put on Fluoxetine, starting with 20mg/day along with bed time diazepam 5mg. He had been drug free for almost 15 days, and prior to this he had been treated with a variety of antidepressants, alone and in combination with other psychopharmacological agents. He seemed to have been unresponsive to imipramine, amitriptyline, nitroxazepine given along with lithium, low doses of haloperidol and thiroidazine, in different combinations. However not all drugs were prescribed in adequate doses and for sufficient length of time to actually call him resistant to treatment with these compounds. He was prescribed fluoxetine in view of its established efficacy in OCD and also in depressive illness.

Two weeks later when he came for his scheduled follow up, he was not taking fluoxetine, as he reported to have developed marked restlessness, anxiety, insomnia, inability to sit still on the 5th day of medication with 20mg/day morning dose of fluoxetine. He was prescribed phenargan 25 mg thrice a day by a local psychiatrist in his city, whom he contacted with these complaints, and he felt relieved of these symptoms within two days.

On this follow up visit he had no such complaints and he had been off medicines for almost 8 days. He was reassured and advised to restart fluoxetine 20mg/day, considering his complaints to be temporary side-effects with the drug, which would pass off after a few days. On the 6th day, he was back in the hospital, very anxious, restless, jittery, and unable to keep his legs still, and unable to sit still, with an urge to move about with which he felt better. The subjective experience and his appearance suggested the possibility of akathisia which is not mentioned as a side-effect in the product monograph of fluoxetine, but anxiety, nervousness and insomnia is reported in about 10%-15% of treated patients (Stark et al., 1985). There was no sign of parkinsonism or dystonia or any other finding on examination.
He was then advised diazepam 10mg/day along with propranolol 40mg/day additionally, both in three divided doses, with which he felt better around 2nd & 3rd day, with symptoms reappearing on discontinuation of these. He was later shifted to imipramine therapy and fluoxetine was totally discontinued because the patient refused to continue to take it.

DISCUSSION

Review of the literature on fluoxetine brought to light a paper by Lipinski et al. (1989), reporting 5 cases of akathisia induced by fluoxetine. Three out of these five cases developed akathisia on the 5th day on 60 mg/day of fluoxetine, one within 12 hours on 20mg/day and the 5th after hours on a dose of 40mg/day.

Fluoxetine induced akathisia is indistinguishable from neuroleptic akathisia, but is milder, and usually comes around the 5th day. Tolerance may develop to it, but it is relieved by either decreasing the dose of fluoxetine or administration of propranolol, an adnergic receptor antagonist. Akathisia in these cases is not associated with signs and symptoms of parkinsonism or dystonia, which almost always accompany neuroleptic induced akathisia. Figures of its exact incidence have to be worked out but is estimated to be between 9.8% - 25% (Lipinski et al., 1989).

The tricyclic induced jitteriness syndrome is identical to the syndrome produced by fluoxetine and both are clinically identical to akathisia (Zubenko et al., 1987). The case history reported suggests akathisia as a side-effect of fluoxetine, since the patient was drug free for over 2 weeks before institution of fluoxetine which was not combined with any other drug likely to produce akathisia.

Lipinski et al., have suggested the probable patho-physiology of fluoxetine induced akathisia. Akathisia follows the inhibition of dopaminergic neurone in the Ventral tegmental area (VTA). Fluoxetine and other compounds that enhance serotonergic transmission (including Tricyclic anti-depressants (TCA’S) and Mono-amine Oxidase Inhibitor’s (MAOI’S)) produce akathisia, by enhancing serotonergically mediated inhibition of Dopamine (DA) cells in the VTA, producing DA deficiency and thus akathisia (Lipinski et al., 1989).

The case report is relevant because of the recent introduction of fluoxetine in the Indian market. Combination of fluoxetine with a neuroleptic compound may increase the risk of developing akathisia with fluoxetine. Moreover controlled prospective studies to determine its true incidence can be carried out.

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