INTRODUCTION
Citalopram is a typical drug of SSRI used in serotonergic dysfunction related disorders, including depression, anxiety, panic disorders, obsessive-compulsive disorder and premenstrual dysphoria (Pollock 2001). Besides headache, tremor is considered as the second most common neurological adverse effect of selective serotonin reuptake inhibitors (SSRIs). Abnormal movements such as acute dystonia, dyskinesias, akathisia, Parkinsonism, exacerbations of Parkinson’s disease, and possibly the neuroleptic malignant syndrome have been associated with the usage of SSRIs. There are citalopram induced akathisia, jaw tremor, bruxism, serotonin syndrome, and dystonic rabbit syndrome case reports in the literature (Najjar and Price 2004, Arshaduddin et al 2004, Parvin and Swartz 2005). We report of a case with titubation and essential tremor strongly associated with citalopram usage.

CASE
A 26 years-old medical student admitted to our clinic for head tremor after citalopram usage for depression. He presented to the outpatient adolescent psychiatric unit with depressive mood and was treated with citalopram 20 mg/day. Two weeks later developed head tremor and postural tremor in the upper extremities. Family history for tremor and other movement diseases was negative. His medical history unremarkable, and there was no history of any neurological illness or extrapyramidal symptoms. He had no history of neuroleptic or other neuropsychiatric therapy and alcohol intake.

Systemic examination, blood pressure, and heart rate were normal. On neurologic examination sensati-
on, motor power, and deep tendon reflexes were normal in all extremities. Cranial nerve examination was unremarkable. There was no rigidity. Tongue or palatal tremor has not been seen. However, on stretching his arms, rhythmic, 7-8 Hz hand tremor was noted bilaterally upper extremities. The tremor was accentuated by anxiety and stress. Also he has titubation.

Laboratory examination revealed normal hemoglobin, red, white blood cells, platelet and differential count, blood glucose, liver and kidney function tests, serum electrolytes, coeruloplasmin and serum copper content. The erythrocyte sedimentation rate, serum protein and electrophoresis were normal. The thyroid function tests (T3 - triiodothyronine, T4 - thyroxine) were within normal limits. Cranial magnetic resonance imaging was normal. Citalopram treatment was stopped, and no other treatment was instituted. After cessation of citalopram there was no deterioration in view of the psychiatric and neurological sense. One week after the cessation of the citalopram both titubation and postural tremor completely disappeared.

DISCUSSION

Citalopram, a potent and the most selective SSRI available, is a widely used antidepressant. Basically, serotonergic projections inhibit dopamine function by means both inhibition of firing of the dopamine cells and inhibition of synaptic release and, probably, synthesis of dopamine in the midbrain, striatum and cortex. Several other observations suggest that dopamine and serotonin systems modulate each other to act in a co-operative manner in the forebrain (Guan and McBride 1989, Ferre et al 1994).

There is a central oscillator at olivocerebellar-thalamocortical-spinal level acting as the primary generator of essential tremor, which is regulated by peripheral component. It is believed that the involvement of serotonin in modulation of olivary excitability by allowing the membrane potential of olivary neurons to be maintained with a narrow range so as to prevent them from generating uncontrollable rhythmic firing (Barragan et al 1985). Although serotonin performs in an excitatory effect within the inferior olive, the loss of a tonically excitatory input may bias olivary neurons toward hyperpolarization, thereby increasing their probability of entering into oscillation in response to synaptic input (Sugihara et al 1995).

The drugs decreasing levels of the serotonin in the inferior olive may spoil the motor system towards uncontrolled oscillations causing tremors and as a result serotonin hyperstimulation occurs (Barragan et al 1985).

Above mentioned mechanism probable explains the etiology of the tremor due to citalopram.

Abnormal movements such as jaw tremor, bruxism, dystonia, deterioration of parkinsonian tremor after citalopram treatment reported. But there is no essential tremor or titubation after citalopram treatment.

CONCLUSION

Citalopram must be taken in mind for causing or aggravating abnormal movement, both essential tremor and titubation may be explained by inhibitory impact on central dopaminergic activity.

REFERENCES


