Dear Editor:

We would like to report a child who experienced tics while she was on fluoxetine.

The patient is a 10-year-old school-girl. She was diagnosed with obsessive–compulsive disorder (OCD) according to DSM-IV-TR criteria through psychiatric examination and tests. According to the information obtained from her mother, she suffered from obsessions for the last five years, which occurred more frequently in the last six months. Fluoxetine 10 mg/d was commenced and behavioral interventions were made for her obsessions. The patient had not been exposed to any other pharmacologic agents prior to this treatment. She was invited to a control meeting one month after her first visit to our clinic, but she and her parents could come two months later. In the control meeting, she stated that her obsessions were decreased in intensity but at the tenth day of the treatment, she had tics in the form of winking, arm movements and voices. These tics were more frequent at the beginning but they decreased in time. Laboratory findings (complete blood count, biochemical analysis, thyroid function tests and ASO) revealed no abnormality. The parents of the patient were informed that the tics may be related to the medical treatment and may be transient. As tics were decreased and the patient benefited from treatment, the patient's mother refused an alternative treatment. During eight months of the follow-up, her medical treatment, accompanied by tics of winking and arm movements at non-disturbing level, was continued. These rare and mild tics disappeared completely at the second week of the cessation of the medicine. The patient has been followed for seven months after the cessation of the drug without any medication, and she no longer had any tics during this period.

It is the suggestion of this report that that tics may be a side effect of fluoxetine in some patients and might indicate a causal relationship. Although tic symptoms related to fluoxetine have been reported previously in adults and adolescents, to the best of our knowledge, no case was reported among children. Extrapyramidal symptoms (EPSs) are uncommon side effects of selective serotonin reuptake inhibitors (SSRIs). The most common EPS associated with SSRIs seems to be akathisia, followed by dystonia and parkinsonism. Fluoxetine is an SSRI which is most associated with extrapyramidal reactions in the majority of cases (Coulter and Pillans 1995, Leo 1996). One plausible explanation for the extrapyramidal symptoms observed in these patients is that serotonin modulates dopaminergic neurons.

Increase in tics have been reported following withdrawal of neuroleptics or following exposure to agents that increase central dopaminergic activity such as L-dopa and CNS stimulants, including cocaine. Using imaging techniques, a number of investigators have also found increased levels of dopaminergic innervation of the striatum in Tourette Syndrome (TS) subjects compared with controls (Albin et al. 2003, Cheon et al. 2004).

SSRI induced tics are one of the rare adverse effects reported in treatment with fluoxetine, sertraline and escitalopram (Eisenhauer and Jermain 1993, Hauser and Zesiewicz 1995, Altindag et al. 2005). There is a case report on the emergence of tics associated with escitalopram and sertraline treatment (Hauser and Zesiewicz 1995), in which the patient, who suffered from panic disorder, experienced involuntary, paroxysmal contractions of the muscles around her right eye and forehead eight weeks after starting escitalopram. Escitalopram was then replaced with sertraline after about 2 months. Tics were reexperienced three weeks after the resumption of sertraline.

Pathological mechanism of tics due SSRIs has yet to be fully elucidated. The interactions between serotonergic and dopaminergic neurotransmitter systems have been suggested
to account for this. (Schillevoort et al. 2002). Ascending serotonergic projections from the dorsal raphe have been invoked as playing part in the pathophysiology of both TS and OCD. This explanation is the most compelling evidence related to OCD and is based largely on the well-established efficacy of potent SSRIs such as fluoxetine in the treatment of OCD. Some investigators have reported that the SSRIs are less effective in treating tic-related OCD than other forms of OCD (McDougle et al. 1993). Additionally, children are 5–12 times more likely to have a tic disorder than adults (Burd et al. 1986). Further research is needed to clarify the exact pathophysiologic mechanism of this adverse reaction.

The most important limitation of our case is that comorbidity of tics and obsessive compulsive symptoms is common. Therefore, the patient should be followed up for a long time to determine whether tics will recur without medication.

REFERENCES


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