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Fluoxetine-induced extrapyramidal symptoms in an adolescent: a case report

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Summary

We present a 15-year-old girl with depression, an obsessive compulsive disorder and conduct disorder, who developed EPS (torticollis, bradykinesia and cogwheel rigidity) while on fluoxetine. No other cause of EPS was present. The patient responded well to benztropine but re-experienced EPS when benztropine was stopped. Fluoxetine and benztropine were used concomitantly for 2½ months and the patient has been off medication for 2 months without EPS. This case report shows that EPS can and does occur in youth with SSRI. Clinicians should be aware of the SSRIs as a potential causative factor for EPS.

Key words: selective serotonin reuptake inhibitor; fluoxetine; children; extrapyramidal symptom

Introduction

As in adults, selective serotonin reuptake inhibitors (SSRIs) have been increasingly used in the treatment of children and adolescents [1, 2]. Because of this wide use even low-frequency adverse effects are becoming more prevalent [3], and reports of extrapyramidal symptoms (e.g. dystonia, dyskinesia, akathisia, parkinsonism and neuroleptic malignant syndrome) associated with SSRI use have been accumulating in the literature [3, 4]. Epidemiological studies suggest that EPS occur in about 1 per 1000 adult patients treated with SSRIs [5, 6].

A literature review on children and adolescents revealed only one EPS report in which paroxetine-induced oculogyric crisis was described in a 9-year-old girl with compulsivity and attention-deficit hyperactivity disorder [7]. Symptoms of ocular pain and a sustained upward gaze occurred approximately 3 days after the addition of paroxetine 10 mg/day to a regimen of pimozide [7].

In this report we present a 15-year-old girl who developed EPS while using fluoxetine. We discuss the clinical features, aetiology and significance of this rare clinical condition.

Case report

"A" was a 15-year-old female in whom a major depressive disorder, OCD (washing, cleaning and checking) and CD according to DSM-IV were diagnosed at age 14 [8]. There was no family neuropsychiatric history and initial medical work-up including whole blood count, blood chemistry panel, liver function tests, ECG, thyroid function tests and child neurology consult revealed no significance.

Abbreviations

EPS	Extrapyramidal symptoms
SSRI	Selective serotonin reuptake inhibitor
OCD	Obsessive compulsive disorder
CD	Conduct disorder
PD	Parkinson's disease

Fluoxetine 20 mg PO qd was started and then increased to 40 mg in six weeks due to slow response to the treatment. We also used 25–50 mg/day quatiapine (an atypical serotonin-dopamine receptor antagonist) between 2½ and 8½ months of fluoxetine treatment when OCD symptoms markedly diminished and CD symptoms were improved. After three more months of 20 mg/day fluoxetine monotherapy (after 12½ months of fluoxetine introduction) A developed "cogwheel rigidity", "bradykinesia", and "episodic torticollis lasting 5–10 minutes three to four times a day". A was seen with torticollis, which looked similar to that seen in patients on typical antipsychotics. The physical and neurological examination was normal except for "cogwheel rigidity and bradikinesia". No physical findings or abnormal movements were obseved in A. Benztropine 2 mg IV resulted in rapid relief of

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torticollis. Whole blood count, blood chemistry panel, liver function tests, ECG and creatine phosphokinase level were normal.

Given the effectiveness of fluoxetine treatment, A and A's parents were unwilling to stop fluoxetine, and benztropine 1 mg PO tid was prescribed along with fluoxetine. A received no EPS for 10 days and benztropine was gradually withdrawn over a week. She re-experienced mild bradykinesia and cogwheel rigidity, but no torticollis, one day after cessation of benztropine. Benztropine 1 mg PO tid was restarted and EPS were resolved within two days. Benztropine and fluoxetine were used for a further 2 months. A has not experienced EPS since cessation of medication 2 months ago.

Discussion

Given the absence of a drug-related cause for EPS (quatiapine poses a relatively low risk for EPS and our patient had not taken quatiapine for three months) [4] and the rapid and time-related response to benztropine, we conclude that A experienced parkinsonian symptoms (bradykinesia and cogwheel rigidity) and an acute dystonic reaction (episodic torticollis) related to fluoxetine. This case report adds to the accumulating literature and is evidence that EPS can and do occur in young patients on SSRI.

The majority of SSRI-related reactions occurred within the first month of treatment, but they have also been reported to occur within a number of months [4]. Our case was a female adolescent on a long-term moderate fluoxetine dose without rapid dose adjustment. The literature offers no supporting evidence for a consistent risk factor, although total daily dose of SSRI, rapid dose escalations, increased age, female gender, concurrent psychotropics known to precipitate EPS, and concurrent disease states such as Parkinson's disease are cited [3–6].

As in our report, fluoxetine is the biggest offender in the literature on adults and acute dystonia is the most frequent EPS finding [3, 4, 9]. EPS is usually reversible and conveniently managed by discontinuing the responsible agent, lowering its dose or using drugs similar to those employed in neuroleptic-induced EPS [9]. On the other hand, co-prescribing of SSRIs and neuroleptics may increase neuroleptic plasma levels (due to P-450.2D6 system blockage), increasing the likelihood of EPS [10, 11].

Conclusions

Clinicians should be aware of the SSRIs as a potential causative factor for EPS. Greater awareness of this potential role could lead to more frequent recognition and help to decrease morbidity. The emergence of SSRI-induced EPS in children and adolescents should be the subject of further research in large clinical samples.

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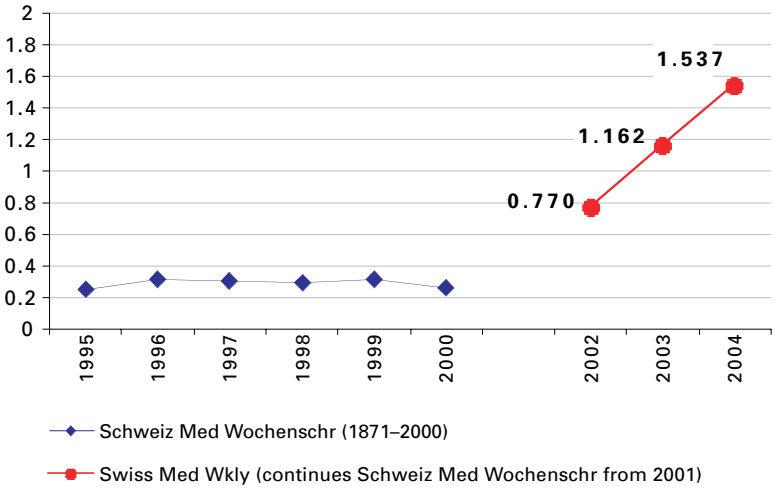

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