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Research Article

Escitalopram Induced Dystonia: A Case Report

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ABSTRACT

Presently a large number of antidepressants are available for the management of depression. Escitalopram is considered to be the safest amongst the various selective serotonin reuptake inhibitors (SSRIs) group of antidepressants. It has been used in depression with a relatively good safety profile. We report here a case of dystonia that developed in patient who was being treated with Escitalopram.

Key words: Escitalopram, dystonia, SSRIs.

INTRODUCTION

One of the earliest reports linking extrapyramidal reactions to SSRIs was published almost 4 decades ago. It was a case of fluoxetine-induced dystonic reaction and parkinsonian rigidity.[1] The authors postulated that the increase in 5-hydroxytryptamine (5-HT) activity resulting from 5-HT reuptake blockade by fluoxetine inhibited both the nigrostriatal and tuberoinfundibular dopaminergic neurons. This has been supported by various studies.[2] It appears that the SSRIs are more frequently associated with these adverse effects than are other antidepressants.[3] Several reviews of case reports of EPS ascribed to the use of SSRIs found that the most common type of extrapyramidal symptom was akathisia, followed by dystonic reactions, parkinsonism, and tardive dyskinesia–like states.[4] Fluoxetine, the most commonly prescribed SSRI during the early 1990s, was implicated in the majority of cases of SSRI-induced extrapyramidal symptoms.[5] Here we report dystonia that developed in a patient treated with Escitalopram.

CASE REPORT

A 32 year old right handed male, Hindi speaking, Muslim by religion, with an education studies upto 7th standard, currently unemployed and the youngest amongst 4 siblings, resident of Kurla was brought by his family members with chief complaints of deviation of mouth to one side after an increase in the dose of the medications he was taking.

The patient was apparently alright till he was 16 years of age, when he started getting generalized tonic clonic seizure episodes. Seizures used to occur independent of fever and any head injury. The patient would have loss of consciousness lasting upto 10minutes, rigidity of the limbs, uprolling of eyeballs, clenched teeth and occasional tongue bite with frothing at the mouth. Computed tomography of the brain revealed no abnormality. His EEG showed generalized epileptiform activity. The patient was started on sodium phenytoin 300mg per day in divided doses.

The seizures were uncontrolled despite medication. The patient would have seizures at his workplace often and he was fired from his job due to health reasons. He became dependent on family members. He started remaining withdrawn, showed decreased interaction with family members, decreased interest in daily and previously pleasurable activities, difficulty falling asleep, experienced fatigue on getting up in the morning was tired throughout the day. Ideas of helplessness, hopelessness and worthlessness were present. His dose of Sodium phenytoin was stepped up to 600mg per day and Sodium Valproate 400mg per day in divided doses was started as seizures were not controlled with a single drug. Keeping in mind the depressive features, the patient was started on Escitalopram 10mg per day. The patient was seizure free and showed an 80% reduction in depressive features with these medications. A month after being seizure free, the seizures restarted. EEG showed epileptiform activity again. Sodium valproate was raised to 900mg per day and seizure frequency reduced until finally it stopped.

The patient started complaining of depressive symptoms yet again and his Escitalopram dose was increased to 15mg per day. He then presented the next day to the emergency department in the evening with protrusion of the tongue outside the mouth which couldn't be taken in with voluntary control, this was continuous and painful after sometime. These symptoms occurred within an hour of consuming Escitalopram. He was given an injection Promethazine and the symptoms were relieved instantaneously. The dose of Ecscitalopram was reduced to 5mg per day and the patient started on Trihexyphenidyl 2mg twice a day. Amitryptiline 50mg per day was started to control the depressive symptoms and gradually the dose of Escitalopram was tapered off. The patient is currently maintained on the medications.

DISCUSSION

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The data for escitalopram and citalopram are limited when we search for extrapyramidal reactions. Escitalopram has been implicated in a few cases such as oculogyric dystonic reaction and rabbit syndrome.[6] Escitalopram induced paroxysmal dystonia has also been reported.[7] Researchers have hypothesized a link between antidepressant induced extrapyramidal reactions and the CYP2D6 phenotype.[8] On analysing the risk factors for these reactions during treatment with SSRIs, including the cytochrome P450 enzyme, and serotonin and dopamine transporter and receptor polymorphisms, it was noted that the risk of EPS with SSRIs seemed to increase with advanced age and the presence of the A1 allele of the dopamine D2 receptor (DRD2) gene Taq1A.[9] Though dystonia is a rare side effect of Escitalopram, it is one the clinician must be aware of and be ready to handle when it arises.

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