ORIGINAL RESEARCH PAPER

Psychiatry

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ESCITALOPRAM -INDUCED GALACTORRHOEA

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ABSTRACT

Although SSRIs are capable of producing galactorrhoea as a side effect, few literatures are available in this regard. Escitalopram ,a SSRI which acts by enhancing the serotonin neurotransmitter availability, may also supress dopamine neurotransmission in tuberoinfundbular pathway. This can result in rise of prolactin level and eventually galactorrhoea. The following case report is about a middle aged woman who developed galactorrhoea following treatment with escitalopram, which subsided on withdrawing escitalopram.

INTRODUCTION

Antipsychotics are the drug with which galactorrhoea is encountered as a common side effect [1] , which is due to the D2 receptor blockade in the tuberoinfundibular pathway. Galactorrhoea, an undesirable side effect is rare with SSRI's and only few cases have been reported [2,3,4] , thus more awareness is required , and an suspicion of SSRI induced galactorrhoea should come into the mind of physicians whenever a patient complaints of pain or secretions over the chest region . In this case report we present the case of a 34 year female treated with escitalopram for her generalised anxiety disorder who developed galactorrhoea which remitted on discontinuation of escitalopram.

CASE

Case Report

34 year old house wife, a graduate, living with husband and children in urban background reported with complaints of vague fear in day to day activities of herself and her family members. She couldn`t concentrate on doing household activities. She was worried that something bad might happen to her family members. She felt tired in daytime and difficulty in falling asleep. Frequently she complained of headache and was irritable while doing homework with her kids. Though she finds it unnecessary to worry about trivial events, she couldn't overcome it for 2 months. Gradually she developed palpitation, shortness of breath and inability to relax at rest in next 2 months. There were no associated stressors. Past history, Family history and Past Personal history was insignificant.

No history of thyroid dysfunction, diabetes mellitus and hypertension. Her physical examination and systemic examination were normal. Her mental status examination revealed predominant anxiety features like fidgetiness, cold peripheries and anxious mood. Thought process was preoccupied with vague fear and helplessness from previous treatment by general practitioners. There was no perceptual abnormality. A provisional diagnosis of Generalised Anxiety Disorder was made. Psychological Assessment with Rorschach and Thematic Apperception Test revealed anxiety nature5,6. Symptom Sign Inventory revealed both depressive and anxiety features. HAM-A score was 24 amounting to moderate anxiety 7. HAM-D score was 12 amounting to mild depression [8].

She was treated with Escitalopram 10mg night and Clonazepam 0.25mg in morning and night for 7 months. Later she reported with milk secretion from breasts with drugs. Serum prolactin was found to be 207ng/ml (high) [9]. So Escitalopram was stopped and continued on Clonazepam. Urine pregnancy test was negative. MRI brain was normal. A week later, Galactorrhoea stopped. She was on clonazepam on PRN basis and stopped completely in three months. Serum prolactin level was found to be [8].32ng/ml, three months after stopping Escitalopram. Since there is temporal correlation with starting of escitalopram and development of galactorrhoea , the increase in prolactin levels can only be ascribed as the side effect of escitalopram .

DISCUSSION

With depression as one of the leading psychiatric morbidity , and SSRIs being the choice of antidepressants , clinicians should expect the uncommon side effects and those being extrapyramidal symptoms, hyperprolactinaemia and galactorrhoea . Based on several clinical trials and neurophysiologic data regarding the inhibition of dopamine neurotransmission by antidepressants, one literature[10]classifies all of these SSRI-induced side effects under the class dopamine-dependent side effects.

This case demonstrated the development of galactorrhea while on escitalopram, which remitted within a short period after the discontinuation of escitalopram. This patient, was thoroughly tested inorder to eliminate all the most probable causes of galactorrhea. Hypothyroidism can cause raise in the levels of thyrotropin-releasing hormone, leading to increased prolactin secretion. Renal impairment may cause secondary hyperprolactinemia. During pregnancy, and in breast-feeding, galactorrhea may be a normal finding. The values of urea, creatinine, and thyroid-stimulating hormone were all within normal range, and a urine human chorionic gonadotropin (HCG) test was negative, we were able to eliminate underlying kidney disease, hypothyroidism, or pregnancy as possible causes of galactorrhea. Although a transient surge (17.80±4.65 ng/mL) of prolactin following citalopram administration was found in some human studies,11 as in our case with prolactin level (207 ng/mL) very high is rare.

The mechanism of prolactin release is not completely understood. Dopamine is a potent inhibitor of prolactin release, and thyrotropin releasing hormone and serotonin (5-HT), promotes prolactin release.

The theory behind serotonergic agents producing hyperprolactin emia is unclear. Some studies suggest that 5-HT may stimulate prolactin release directly via postsynaptic 5-HT receptors in the hypothalamus,12 or indirectly via 5-HT mediated inhibition of tubero-infundibular dopaminergic neurons[13] .The interactions between the dopaminergic and serotonergic systems are complex. SSRIs can inhibit dopaminergic neurotransmission not only by their

effects on dopamine secretion or recapture on dopaminergic receptors, but also indirectly through serotonergic mediation.[14] Complex changes of dopaminergic neurotransmiss ion have been established with SSRIs.[15] However, there is little synaptic contact between serotonergic fibres and dopaminergic cells, indicating that if direct inhibition of dopamine cells occurs, it is through volume transmission of serotonin in the region.[16] There is more direct evidence for serotonergic stimulation of GABAergic neurons in the vicinity of tuberoinfundibular dopamine cells, based on the presence of 5-HT1A receptors on these cells.[17] Assuming that these GABAergic cells are interneurons, their 5-HT stimulation by SSRIs would result in inhibition of tuberoinfundibular dopamine cells, releasing the tonic inhibition of prolactin.

Gulsun et al.[18,19] have previously reported two cases with escitalopram-induced galactorrhea with and without prolactine mia. As far as we know this is the first report to show the case with escitalopram-induced galactorrhea and hyperprolactinemia in Korea. While the galactorrhea sometimes seen after treatment with seroton ergic medications appears to be a benign side effect, its occurrence offers new insights into the impact of serotonergic drugs on brain neurotransmitters and hormonal regulation. Clinicians should be aware of the possibility of galactorrhea in this context. Researchers should continue to explore the neurohormo nal effects of serotonergic medications to gain insights into their effects.

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