Case report

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Escitalopram induced Galactorrhea: a Case Series Emphasizing Delayed Onset in Long-Term Therapy

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Keywords

Escitalopram; hyperprolactinemia; galactorrhea; case reports

Abstract

Background: Escitalopram, a widely used psychotropic (SSRI) used for depression, has rarely been associated with hyperprolactinemia and galactorrhea. Current literature at present has isolated case reports, with no prior series documenting this adverse effect after prolonged use. **Aim:** To present four cases of symptomatic hyperprolactinemia and galactorrhea with onset after 2 – 3 years of escitalopram monotherapy. **Case reports:** This case series is a retrospective analysis of four female patients aged between 27 – 58 years presenting to our OPD SKIMS MCH Bemina Srinagar with bilateral galactorrhea under escitalopram treatment (10 – 20 mg/day). Organic causes were ruled out via comprehensive workup from different departments (serum β - hCG, TSH, MRI brain, USG breasts/pelvis). Serum prolactin levels were measured on presentation and after discontinuation of the SSRI. Patients developed galactorrhea after 2 – 3 years of dose range: 10 – 20 mg/day, having prolactin levels that ranged from 202 – 322 ng/mL, in which resolution occurred within 4 – 8 weeks after discontinuation. **Conclusion:** Escitalopram can induce delayed-onset hyperprolactinemia and galactorrhea after prolonged use, even at standard doses. Patients on SSRIs should be evaluated for galactorrhea irrespective of duration and dosage. A strategy should be devised for early pickup.

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Introduction

Escitalopram, a widely selective serotonin reuptake inhibitor (SSRI), is commonly prescribed for depression due to its different aspects (efficacy, safety, and tolerability [1]. Side effects reported are gastrointestinal issues and sexual dysfunctions, whereas endocrine abnormalities such as hyperprolactinemia and galactorrhea are rarely reported. SSRIs may elevate prolactin via a pathway of serotonin (5-HT)-mediated inhibition of dopaminergic neurons in the tuberoinfundibular pathway, reducing dopamine's tonic suppression of prolactin release [2]. However, hyperprolactinemia is well-documented as an adverse effect of antipsychotics [3,4]. Few SSRI-induced galactorrhea cases have been reported, particularly for escitalopram [4]. Few case reports linked to escitalopram induced galactorrhea have been reported [5,6] with no prior case series describing this phenomenon after long-term use. We had patients visiting our outpatient department at SKIMS MCH Bemina Srinagar, who developed symptomatic hyperprolactinemia within the span of 2 – 3 years of escitalopram monotherapy.

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Case reports

Patient 1:

A 27 -year-old unmarried graduate female, urban, with a total duration of recurrent depression of 2.5 years, was doing well on escitalopram. The dose at the time of glactorrhea was 15 mg with no history of medical, surgical, or neurological comorbidities presented in the outpatient department of psychiatry, SKIMS MCH Bemina, with a history of 4 months of bilateral milky nipple discharge. The patient had a first consultation with the department of obstetrics and gynecology, where baseline investigations (CBC, KFT, LFT, TSH, T3, T4,β-HCG, USG abdomen/Pelvis were done, which were within normal limits, ruling out all organic causes. The patient was referred to endocrinology, where MRI Brain and USG bilateral breasts were unremarkable studies, but sr. prolactin levels were Prolactin 202 ng/mL (ref: 3 – 25 ng/mL). Ruling out all causes, the endocrinology department sent the patient again to the department of psychiatry for review. The patient was discussed in a board, and a decision was taken to stop all psychotropics for 4 weeks till repeat serum prolactin is done. The patient again visited our outpatient department, and serum prolactin levels were 10 ng/mL after 4 weeks. Galactorrhea got resolved completely after stopping escitalopram. Patient was managed with CBT and vortoxetine 10mg; no relapse.

Patient 2:

A 32-year-old woman, unmarried, illiterate from tribal rural strata of population, with major depressive disorder, on escitalopram 20mg for 3 years, with a history of non-surgical, medical, or neurological comorbidities. The patient had poor follow-up, and for three years, the patient had continued medication. The patient reported to the outpatient department of psychiatry, presenting with milky discharge from the right breast for 4 months. She had not used any medication for a long period of time, which could lead to galactorrhea. We ordered baseline investigations (CBC, LFT, KFT, USG bilateral breasts and abdomen /pelvis, TFT, B-HCG, chest X-ray, and ECG in all leads) in the outpatient department that were within normal limits. The patient was referred for endocrine workup to SKIMS Soura Department of Endocrinology, where MRI Brain and hormone workup was done, which revealed no abnormal findings in MRI Brain (Figure 2), and serum prolactin was 287 ng/mL; organic causes were ruled out, and the patient was again sent to the Department of Psychiatry. A discussion board was constituted for further course of action. The decision was taken to stop all psychotropic substances for a few weeks, and the patient was put on CBT. The patient reported to OPD after six weeks with a prolactin level of 15ng/ml, which was advised to get done on the next visit. Galactorrhea had resolved. The Patient was put on mirtazapine 15 mg. The patient had no relapse.

Patient 3:

A 45-year-old woman, married with primary infertility (Documented fallopian tube obstruction) with no other history of medical, surgical or neurological illness, belonging to urban sphere with Adjustment disorder with prolonged depressive episode, treated with escitalopram 15 mg for 2 years had approached to primary care physician at sub district level for consultation in view of milky discharge from both breasts for 5 months. The patient was referred to a super-specialty hospital for evaluation. At the medicine outpatient department of su-

Figure 1. T1 flair of 27 year female

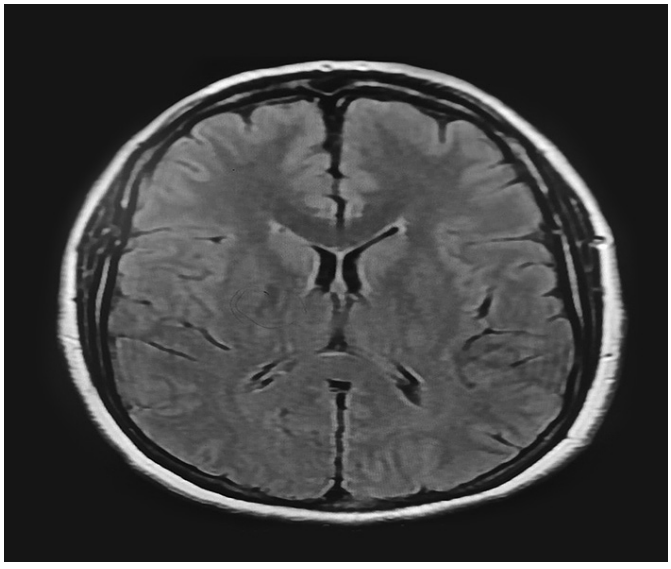


Figure 2. Flair of 32 years old female

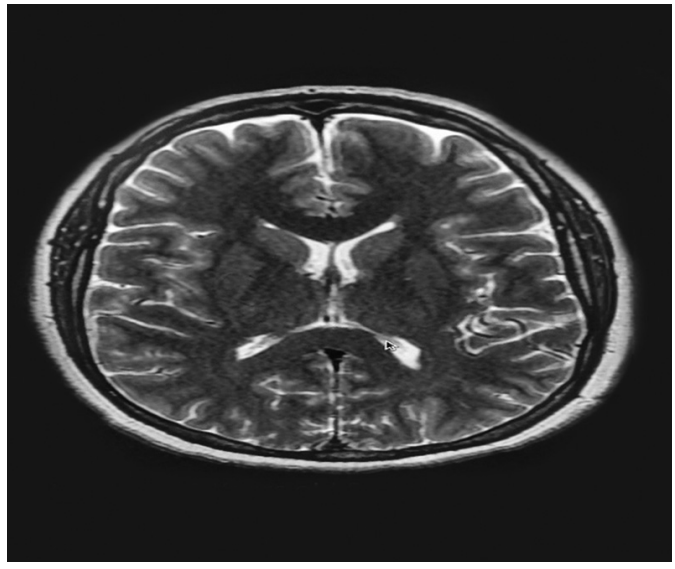


Figure 3. T2 Flair of the 45 year female

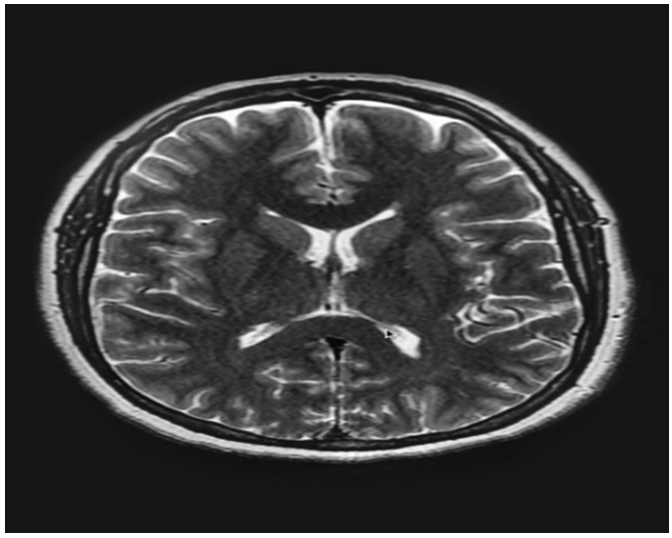
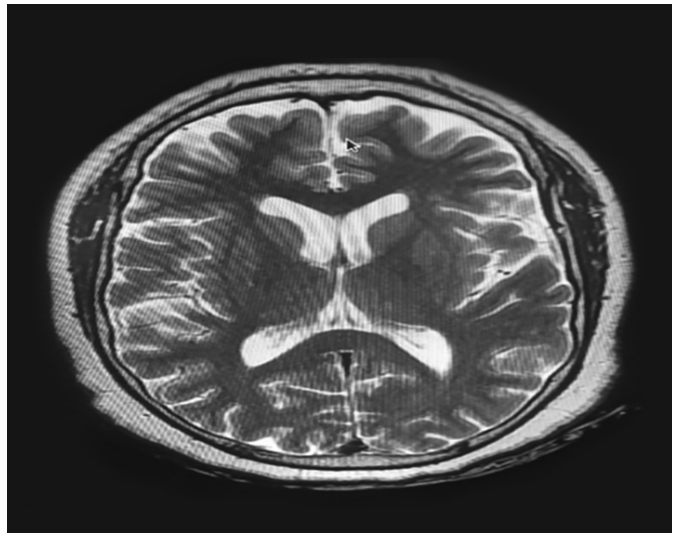


Figure 4. Flair of 58 years female



perspeciality, the baseline investigations (CBC, LFT, KFT, TFT, USG bilateral breasts and abdomen /pelvis chest X-ray, β-hCG, and ECG in all leads) were done, which resulted within normal limits. The patient was subsequently referred to Endocrine OPD, where an MRI Brain and a hormone profile were done. Prolactin was 322 ng/mL; MRI Brain (Figure 3) had nothing significant. After ruling out all organic causes, the patient was referred to the psychiatric OPD. Detailed evaluation revealed that the patient had not taken any medication that could lead to galactorrhea. The case was seen by a consultant psychiatrist in OPD, and a decision was taken to discontinue escitalopram. Patient was put on CBT, and patient was advised to undergo serum prolactin after 4 weeks. Prolactin at 4 weeks was 38 ng/mL (still mildly elevated but decreasing), and galactorrhea improved (mild discharge, occasional now from frequent).On follow-up after 8 weeks, resolution was seen. Patient was put on vortoxetine; no relapse.

Patient 4:

A 58-year-old working woman, married, urban, on escitalopram 15 mg for 2 years, prescribed by her primary physician in view of chest discomfort, sadness of mood, apprehension, came to the psychiatric OPD at SKIMS MCH Bemina in view of having distress due to white milky discharge for the last 3 months from both breasts. The baseline investigations (CBC, LFT, KFT, TFT, USG bilateral breasts and abdomen /pelvis chest X-ray, β-HCG, and ECG in all leads that were within normal limits). The patient was referred to the department of medicine for further management. The patient was evaluated with an MRI Brain (Figure 4) showing no abnormality, and serum Prolactin was 249 ng/mL. All other possibilities were ruled out, and the patient had no history of any drug use that could cause galactorrhea. The patient was sent for review of medication by the Department of Medicine. Escitalopram was discontinued in the case discussion board. The patient was lost to follow-up for the 4-week prolactin test, but she reported resolution of galactorrhea at 6 weeks. Prolactin level at 6 weeks was not measured.

Discussion

We have reported four cases of escitalopram associated galactorrhea. The temporal relationship between escitalopram use and the onset of galactorrhea, with resolution after discontinuation, suggests a causal link. Escitalopram increases serotonin availability by activating 5 - HT_2 receptors in the hypothalamus, thereby suppressing dopamine release from TIDA neurons and disinhibiting pituitary lactotrophs and increasing prolactin secretion our all participants were female patients on long-term escitalopram (2 - 3 years) developing galactorrhea with significantly elevated prolactin (202 - 322

Table 1. Clinical pattern of cases in our study

Feature	Patient 1	Patient 2	Patient 3	Patient 4
Age	27	32	45	58
Escitalopram dose	15 mg	20 mg	15 mg	15 mg
Duration of use	2.5 years	3 years	2 years	2 years
Prolactin level(ng/ml)	202	287	322	Not done
Resolution time	4 weeks	6 weeks	8 weeks	6 weeks

ng/mL) in which resolution occured after discontinuation of escitalopram that is in line with the shim etal [7-10]. The onset of symptoms in our patients after prolonged use (4 months-5 years), dose-independence (occurringinduced at 10 - 20mg), and complete resolution post-cessation align with the study observing that escitalopram-induced galactorrhea can occur at lower doses than previously thought [11]. All of our participants were females who corresponded with higher susceptibility due to estrogen's amplification of prolactin release [12]. The levels of Prolactin normalization from (10 – 38 ng/mL) and symptom resolution took place within 4 – 8 weeks after discontinuation, echoing [9,10].

Our case series indicates that escitalopram can induce symptomatic hyperprolactinemia and galactorrhea, even after prolonged use (2 – 3 years) at standard doses (10 – 20 mg/day). Escitalopram-induced galactorrhea is a consequential adverse effect requiring clinical vigilance. Dominance of females suggests estrogen-mediated susceptibility. There should always be a focus on drug history while evaluating such cases.

Patient consent forms were obtained from all four patients, citing that privacy, confidentiality, and dignity will be maintained. Information about publishing the data or clinical images was also written in the form, and all patients gave their consent. The patients understand that their personal information will not be published or their identity disclosed.

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None.

Conflict of Interest

None to declare.

Funding Sources

None.

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