CASE REPORT

Transient normoprolactinemic galactorrhea induced by fluoxetine

**Fluoksetine bağlı, geçici normoprolaktinemik galaktore**

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**ABSTRACT**

Hormonal side effects of antidepressants are infrequent. Galactorrhea is rarely reported among antidepressant related side effects. Antidepressants can directly stimulate postsynaptic 5-Hydroxytryptamine (5-HT) receptors in the hypothalamus or indirectly inhibit the tuberoinfundibular dopaminergic neurons through 5-HT, which may increase prolactin levels and later cause galactorrhea. However, galactorrhea may develop despite normal prolactin levels during antidepressant treatment. We present a case of normoprolactinemic galactorrhea in a woman, related with fluoxetine treatment. This report highlights the presence of unidentified mechanisms of selective serotonin reuptake inhibitor induced galactorrhea. J Clin Exp Invest 2013; 4 (1): 105-106

**Key words:** Fluoxetine, galactorrhea, side effect

**INTRODUCTION**

Several drugs can cause galactorrhea, and it needs to be differentiated from other local or neuroendocrinological causes. All conventional antipsychotic drugs block D2 receptors on lactotroph cells and thus remove the main inhibitory influence on prolactin secretion.¹ Tricyclic antidepressants and selective serotonin reuptake inhibitors (SSRI) are less frequent causes.²,³ There is evidence that serotonin may stimulate prolactin release directly via postsynaptic 5-Hydroxytryptamine (5-HT) receptors in the hypothalamus,⁴ or indirectly via 5-HT mediated inhibition of tubuloinfundibular dopaminergic neurons.⁵ However, galactorrhea due to antidepressants is not consistently associated with elevated prolactin levels, which may suggest still unexplained mechanisms of antidepressant-induced galactorrhea.⁶,⁷

Herein, we report a case of euprolactinemic galactorrhea in a woman with generalized anxiety disorder while on treatment with fluoxetine.

**CASE**

A 29-year-old woman with two children visited a psychiatric outpatient clinic with complaints of excessive and uncontrollable worry about minor life events, feeling restlessness, irritability, muscle tension, tiring easily, and poor sleep that started 8 months prior to admission. She had no history of endocrine or reproductive pathology or psychiatric problems. She was diagnosed as generalized anxiety disorder according to DSM-IV criteria and was started on fluoxetine 20 mg per day. After 4 weeks of medication, her complaints declined. However, she developed unilateral galactorrhea (the nonpuerperal discharge of milk-containing fluid from the breast). She had no history of galactorrhea, thyroid disease, and polycystic ovary disease. She first noticed the discharge on treatment day 21 and described it as white-creamy and from right nipple. She did not notice any bloody, greenish, or foul-smelling discharge nor report sexual dysfunction. She consulted her gynecologist, who recommend-
ed mammogram and breast ultrasonography. The pregnancy test was negative. The results of these tests and breast examination were normal. Serum prolactin level on treatment day 28 was 18.18 ng/mL (reference range: 2.5-29 ng/mL). Because her galactorrhea developed after initiations of her medication with fluoxetine, her medication was discontinued. Buspirone 5 mg/day treatment was started and gradually raised to 20 mg/d. Eight days after stopping fluoxetine, the patient reported reduction and cessation of galactorrhea. During 3 months of follow-up, the patient maintained well on buspirone and there was no reemergence of galactorrhea.

DISCUSSION

Galactorrhoea caused by the use of fluoxetine has been reported earlier and the commonly perceived cause is hyperprolactinemia.\(^8\) Fluoxetine has been shown to potentiate elevation of prolactin levels inducing other stimuli, including insulin, fenfluramine, and 5-HT.\(^9\) However, hyperprolactinemia is not the only mechanism responsible for the development of SSRI-induced galactorrhoea.\(^6,7\) The exact mechanism of galactorrhoea remains unknown in many cases.

Our patient developed galactorrhoea without hyperprolactinemia after beginning fluoxetine therapy. The strict temporal relationship between the use of the drug and the onset of galactorrhoea, as well as the resolution once treatment was discontinued, suggests a causal link between the two phenomena. Although unilateral galactorrhoea induced by psychotropic medications is rare,\(^10\) our patient developed unilateral galactorrhoea with normal mammogram and breast ultrasonography, indicating a complex relationship between antidepressant use and galactorrhoea.

To the best of our knowledge, we are the first to report an association with fluoxetine use and galactorrhoea without elevated prolactin level. Clinicians should consider fluoxetine as a possible cause of galactorrhoea even with normal prolactin levels. Future research should investigate the exact mechanisms of antidepressant-induced normoprolactinemic galactorrhoea.

REFERENCES