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Letter to the Editor

Paroxetine induced galactorrhoea – A case report

1. Introduction

The selective serotonin reuptake inhibitors (SSRIs) are one of the most commonly used agents to treat panic disorders. Paroxetine is preferred by the clinicians for its calming, sedating and comparatively lesser activating actions in the initial phase of treatment compared to other SSRIs like fluoxetine and sertraline (Cascade et al., 2009).The common side effects of paroxetine are gastrointestinal upset, sexual dysfunction, and prominent withdrawal reaction in the form of akathisia, dizziness, and restlessness upon sudden discontinuation. Galactorrhoea has been only rarely mentioned as a side effect of this drug. However, there are reports of 8-fold higher risk of galactorrhoea upon usage of SSRIs, compared to other antidepressants (Egberts et al.,1997).

Galactorrhoea is defined as discharge of milk or milk like secretions from the breast in the absence of pregnancy or beyond 6 months postpartum. In most pathophysiological states, the final pathway leading to galactorrhoea is an inappropriate release of prolactin. Two mechanisms have been considered to explain prolactin release induced by the serotonergic system: presynaptic inhibition of dopamine discharge by serotonergic receptors or direct stimulation of hypothalamic postsynaptic serotonergic receptors (Bronzo and Stahl., 1993; Egberts et al.,1997). Hyperprolactinemia may be due to pituitary tumors, drugs that inhibit hypothalamic dopamine, hypothyroidism, excessive estrogen intake, stress or hypothalamic lesions. Here, we present the case of a 42-year old woman who was treated with paroxetine for her panic disorder and developed galactorrhoea with hyperprolactinemia that resolved upon discontinuation of the drug.

2. Case report

We report a case of galactorrhoea in a 42- year-old (Mrs.GN) woman having 2 children and getting treatment for panic disorder as an outpatient. For the last 6 months she was treated with escitalopram upto a maximum dose of 20 mg per day with clonazepam 0.5 mg on SOS basis. Due to partial improvement, paroxetine in the controlled release formulation was started at a dose of 12.5 mg, and within 10 days, the dose was increased to 25 mg. Within a month, she showed marked improvement in all the spheres. After about 4 week of continuous treatment with 25 mg paroxetine, the lady noticed milk coming out spontaneously from both the nipples. This patient had no previous history of galactorrhoea. The volume was significant enough that discharge dripped down her abdomen. She did not notice any bloody, greenish, or foul-smelling discharge. The medication was discontinued the next morning, and the discharge ceased on the third day. Paroxetine was substituted with escitalopram on the third day and till date further recurrence of galactorrhoea was not reported. We sought to eliminate the most likely causes of galactorrhoea. The lady was married, having regular periods, and no other abnormality could be found on physical examination.

No evidence of any extrapyramidal symptoms was found. Symptoms suggestive of raised intracranial pressure like headache or visual disturbance were not present, and she had no history of local surgery or herpes zoster infection. Hypothyroidism results in increased levels of thyrotropin-releasing hormone, which increases prolactin secretion. Kidneys clear prolactin, and thus, kidney disease may cause secondary hyperprolactinemia. During pregnancy, and for up to 2 years after cessation of breast-feeding, galactorrhoea may be a normal finding. Because Mrs. GN's routine measurements of urea, creatinine, thyroid-stimulating hormone, and beta human chorionic gonadotropin (b-HCG) were all normal, we were able to eliminate underlying kidney disease, hypothyroidism, or pregnancy as possible causes of galactorrhoea.

Serum prolactin was measured on the day galactorrhoea, and was found to be higher than the normal limits (125.50 ng/mL). Repeat serum prolactin assessment 3 weeks and 8 weeks after discontinuation of paroxetine was within normal limits (32.4 ng/mL and 21.23 ng/mL respectively). Paroxetine was assumed to be responsible for the galactorrhoea and was stopped, following which, the galactorrhoea stopped completely within 3 days. The prolactin level was again found to be normal when assessed 12 weeks after stoppage of galactorrhoea.

3. Discussion

There are sporadic cases of paroxetine induced galactorrhoea in literature (Bonin et al., 1997; González et al., 2000; Morrison et al., 2001; Sertcelik et al., 2012). According to our research the prolactin levels were within normal limits in only two cases (Davenport and Velamoor, 2002; Chakraborty et al., 2010). The relationship between galactorrhoea and serum prolactin levels is not clear. The results of animal and human studies investigating the effects of individual antidepressants on plasma concentrations of prolactin are controversial, showing either normal or elevated levels (Egberts et al., 1997). The approach to patients should comprise discontinuation of the implicated SSRI, careful documentation of the galactorrhoea, and documentation of recent menstrual history. Pregnancy testing and assessment of thyroid status should be done where menstrual history is equivocal or hypothyroidism is a possibility. Assessing prolactin level should be undertaken where clinically indicated or where there is significant patient anxiety. In cases of non-resolving galactorrhoea, clinicians should direct their attention to neoplastic, structural, metabolic, and other causes, as described in Pena and Rosenfeld's recent comprehensive review (Pena and Rosenfeld, 2001). The exact mechanism of galactorrhoea remains unknown in many cases. Hence more research is required to understand the true mechanism behind SSRI-induced galactorrhoea. Hence the clinicians should be aware that galactorrhoea can occur as a side effect of SSRI and may require stoppage of these otherwise necessary drugs in these patients.

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