Ziprasidone-induced galactorrhea: A case report

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Abstract

OBJECTIVES: Second-generation antipsychotics presumably lack the typical side effects of conventional antipsychotics.

METHODOLOGY: A 34 year old Caucasian woman with ICD-10 diagnosis of Recurrent depressive disorder with current moderate symptoms, and with a history of repeated self-injury was treated with lithium, clonazepam and ziprasidone.

RESULTS: On the ninth day of ziprasidone administration, galactorrhea appeared. After 36 days of ziprasidone therapy, galactorrhea persisted. The prolactin plasma level was 28 ng/ml. Thyroid tests (TSH,T3,T4) and the lithium plasma level were within the normal range during ziprasidone treatment. Two weeks after the ziprasidone withdrawal, galactorrhea disappeared and the prolactin level decreased down to 18 ng/ml.

CONCLUSION: Psychiatrists should be aware that even second-generation antipsychotics, including ziprasidone, have a propensity to cause side-effects associated with the dopamine D2 receptor blockade, such as galactorrhea.

1. Introduction

Second-generation antipsychotics (SGA) presumably lack the typical side effects of conventional antipsychotics. Most of these side effects may result in severe impairments of a patient’s physical condition. For example, in women longstanding hyperprolactinemia can lead to gynaecomastia, oligomenorrhea, amenorrhea, galactorrhea, cessation of normal cyclic ovarian function, loss of libido, hirsutism or increased long-term risk of osteoporosis [10]. However, increase of prolactin levels has been
reported in some SGA, as well: amisulpride [2,6], risperidone [5], or olanzapine [7,8]. We present a case report of galactorrhea following treatment with ziprasidone.

2. Case report

A 34 year old Caucasian woman with ICD-10 diagnosis of Recurrent depressive disorder with current moderate symptoms, and with a history of repeated self-injury was admitted to the Psychiatric Center Prague. She was treated with lithium up to 1200 mg per day (p.d.), clonazepam 1.5 mg p.d., and ziprasidone 80 mg p.d. On the ninth day of ziprasidone administration, galactorrhea appeared. The patient’s previous antipsychotic medication, 50 mg of sulpiride daily, was stopped 4 days prior to the ziprasidone treatment and was not associated with any endocrine abnormalities. After 36 days of ziprasidone therapy, galactorrhea persisted. Prolactin levels were determined by the Microparticle Enzyme Immunoassay on 36th day of ziprasidone treatment and two weeks after the ziprasidone withdrawal. The sensitivity of the assay is 0.6 ng/ml. The normal range established for this assay is within 1.39–24.2 ng/ml. The prolactin plasma level was 28 ng/ml. Thyroid tests (TSH, T3, T4) and the lithium plasma levels (0.45–1.01 mmol/l) were within the normal range during ziprasidone treatment. Two weeks after the ziprasidone withdrawal, galactorrhea disappeared and the prolactin plasma level decreased down to 18 ng/ml.

3. Discussion

The are data indicating that ziprasidone can cause a marginal and transient elevation of prolactin levels [9], and thus it has the potential to induce galactorrhea. We observed galactorrhea and hyperprolactinaemia after ziprasidone and concomitant administration of lithium and clonazepam with consequent disappearance of the side effects following ziprasidone withdrawal. Lithium is not generally associated with a risk of prolactin elevation; also, co-medication with ziprasidone is considered as safe [1]. In addition, animal data showed that administration of clonazepam even inhibits prolactin release [3]. To our knowledge, this is the second published report on galactorrhea following treatment with ziprasidone [4], first in an adult patient. Psychiatrists should be aware that even novel SGA, including ziprasidone, have a propensity to cause side-effects associated with the dopamine D2 receptor blockade, such as galactorrhea.

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REFERENCES