

Introduction: Amenorrhea secondary to hyperprolactinemia is one of the frequent adverse effects associated with the use of atypical antipsychotics. It is often neglected but can interrupt the compliance of treatment. Several studies indicate that olanzapine does not significantly affect serum prolactin levels in the long term, although contrary has been observed in few case reports.

Objectives: To report a case of olanzapine-induced amenorrhea due to hyperprolactinemia.

Methods: A 27-year-old woman with history of stillbirth 5 months prior, presented to OPD with hallucinatory behaviour and socio-occupational dysfunction for 5 months. She was on tianeptine 12.5 mg, escitalopram 10 mg and alprazolam 0.5 mg at presentation and was having regular menses. On assessment, she was diagnosed with unspecified psychosis. Her ongoing medications were stopped and she was started on Olanzapine (optimized to 20 mg/day) after which she reported significant improvement however developed amenorrhea within next 2 months hence advised to consult Obgyn. Urine pregnancy test came out negative and prolactin level was found to be 64.2 ng/ml. Other investigations including MRI were within normal limit. Olanzapine was cross tapered with Aripiprazole (maintained at 10 mg/day). Clonazepam was advised SOS for anxiety.

Results: After 1 month of aripiprazole treatment, monthly menses resumed and prolactin level returned to normal range. No biological dysfunction or other side effects were reported by the patient.

Conclusions: Olanzapine-induced amenorrhea secondary to hyperprolactinemia, is a rare but possible event. We report a case in which olanzapine induced amenorrhea normalized after switching to aripiprazole. Baseline prolactin level should be obtained as they help in the management of patients with neuroleptic-induced hyperprolactinemia.

Disclosure: No significant relationships.

Keywords: Olanzapine; Hyperprolactinemia; Amenorrhea; Aripiprazole

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Hipersexuality in aripiprazole treatment : A case report

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Introduction: Aripiprazole is an antipsychotic that differs from the group in that it is a partial agonist of the D2 receptor, being also a partial agonist of 5HT1A and an antagonist of 5HT2A. Most antipsychotics cause a decrease in libido, affecting both sexual desire and function. According to the literature, D2 partial agonists can cause the appearance of compulsive behaviors as an adverse effect in 6-24% of patients. Among these behaviors you can find hypersexuality. In most cases, it subsides when treatment is stopped. We describe the case of a patient with bipolar disorder who develops hypersexual behaviors following the aripiprazole treatment. This is a 61-year-old bipolar patient receiving valproate and risperidone. It requires hospital admission due to manic symptoms where dysfunctional tremor is observed. Change from risperidone to aripiprazole. Subsequently, hypersexual behaviors appear, increased

libido, obsession with sexual activities (compulsive masturbation with TV programs, mobile applications, cartoons) as well as delusional ideas about “receiving sexual gazes” with no other manic symptoms.

Objectives: To determine the possibility that hypersexuality was induced by treatment with aripiprazole.

Methods: The appearance in the time line of hypersexuality after the change of treatment would be indicative of causality.

Results: After switching back to risperidone, compulsive sexual behaviors disappear but not the delusional idea of being the focus of sexual gazes by everyone.

Conclusions: Although it is not a common adverse effect, hypersexuality is listed in the literature as a rare adverse effect.

Disclosure: No significant relationships.

Keywords: hipersexuality; D2partialagonist

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Syndrome of Irreversible Lithium-Effectuated Neurotoxicity (SILENT): A Review

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Introduction: Lithium has a narrow therapeutic window. Frequent monitoring of both serum levels and clinical signs of toxicity is warranted because toxicity may be present even when concentrations are within the therapeutic range. Persistent neurological signs and symptoms of lithium intoxication gained clinical attention in the 1980s and were named Syndrome of Irreversible Lithium-Effectuated Neurotoxicity (SILENT).

Objectives: To review the long-term neurological sequelae of lithium intoxication (SILENT) to highlight their clinical presentation, assessment, management and preventive measures.

Methods: Non-systematic review of literature through search on *PubMed/MEDLINE* for publications up to 2021, following the terms *syndrome of irreversible lithium-effectuated neurotoxicity*.

Results: Neurological manifestations of lithium poisoning may persist, even after effective removal of the drug – SILENT. The most frequent sequelae are cerebellum and brain stem dysfunction, extrapyramidal symptoms and dementia. They may last for weeks, months or years. Infection, dehydration, deteriorating renal function or the addition of other drugs may precipitate acute toxicity. Irreversible damage is difficult to treat. Some cases show spontaneous recovery that may be total, but in others, sequelae persist. Helpful measures include the avoidance of acute intoxications with lithium, long-term and continuous dose adjustment and serum level monitoring, stricter exclusion criteria for starting lithium, and aggressive treatment of acute neurotoxicity. Once the long-term neurologic sequelae have set in, the patient should be managed according to the impediment (physical rehabilitation, speech, cognitive training).

Conclusions: It is important to raise the awareness of SILENT so that clinicians are able to avoid it. There should be a low threshold for suspecting the existence of toxicity.

Disclosure: No significant relationships.

Keywords: Lithium; psychopharmacology; toxicity; psychotropics