

A Case Report on Amisulpride-Induced Hyperprolactinemia Presenting as Secondary Amenorrhea

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Abstract

Amisulpride, an atypical antipsychotic, a selective dopamine D2/ D3 receptor antagonist, is commonly used for treating psychosis and mood disorders. However, one of its known adverse effects is hyperprolactinemia due to dopamine blockade in the tuberoinfundibular pathway. We report the case of a 41-year-old woman who developed secondary amenorrhea following initiation of amisulpride. Her investigations showed a marked increase in prolactin level. Her symptoms improved after discontinuing amisulpride. This case highlights the importance of recognizing medication-induced endocrine dysfunction.

Keywords: Hyperprolactinemia, Amisulpride, Amenorrhea, Prolactin.

Introduction

Hyperprolactinemia is a common endocrine disorder which can be usually associated with prolactinomas, hypothyroidism, or drug use. Prolactin levels can increase as a result of antipsychotics, especially those that block dopamine D2 receptors, which can interfere with dopamine's inhibitory effect on prolactin release. [1] Even low doses of amisulpride can raise blood prolactin levels in a dose-dependent manner, even though it is an effective antipsychotic agent. Women become more affected because of the various consequences of high levels of prolactin levels such as infertility, galactorrhea, amenorrhea, and bone demineralization.[2]

Case Presentation

A 41-year-old lady, who was a teacher, married with two children, was referred from the Gynecology Department for evaluation of hyperprolactinemia. This was found on evaluation of amenorrhea of four months duration.

She was known to have bipolar disorder and was on follow-up with psychiatry. She was being managed with olanzapine 5 mg and venlafaxine 50 mg, for a long time. She had tolerated both these medications well for several years. As she had residual symptoms, amisulpride 50 mg was added to her regimen in November 2024. She developed Menstrual irregularities within a few weeks of starting amisulpride. Later

this progressed to amenorrhea over time. There was no evidence of galactorrhea, visual disturbances, or headaches. Clinical examination revealed normal secondary sexual characteristics, with no thyroid enlargement or other abnormalities. She had stable vital signs. There was no history suggestive of PCOS or pituitary pathology.

Laboratory tests showed increased serum total prolactin of 2225.06 $\mu\text{IU/mL}$ (Pre-menopausal range: 71.1–568.5 $\mu\text{IU/mL}$), and normal TSH: 3.68 $\mu\text{IU/mL}$, FSH and other pituitary hormones were within normal limits. MRI brain and pituitary was not done at this stage due to the high clinical suspicion of drug-induced etiology. Amisulpride was promptly discontinued. Repeated prolactin after 3 weeks of stopping amisulpride showed a decline. total prolactin to 166.55 $\mu\text{IU/mL}$, Free Prolactin: 34.28 $\mu\text{IU/mL}$ and PEG Precipitation Ratio showed 79.42% (suggestive of monomeric prolactin dominance). These findings indicated a strong link between the patient's symptoms and amisulpride use.

Table 1: Clinical Timeline

Date/Period	Clinical Event
Before Nov 2024	Patient stable on olanzapine and venlafaxine; regular menstrual cycles.
Nov 2024	Amisulpride 50 mg started for residual depressive symptoms.
Dec 2024–18 Feb 2025	Patient developed amenorrhea; prolactin measured at 2225.06 $\mu\text{IU/mL}$.
26 Feb 2025	Amisulpride stopped under supervision.
18 Mar 2025	Repeat prolactin dropped to 166.55 $\mu\text{IU/mL}$; menstruation resumed.

The clinical course and therapeutic milestones are summarized in Table 1

Table 2: Hormonal Profile Summary

Test	Result	Normal Range
Total Prolactin (initial)	2225.06 $\mu\text{IU/mL}$	71.1–568.5 $\mu\text{IU/mL}$
Total Prolactin (after 3 weeks)	166.55 $\mu\text{IU/mL}$	71.1–568.5 $\mu\text{IU/mL}$
Free Prolactin	34.28 $\mu\text{IU/mL}$	N/A
PEG Precipitation Ratio	79.42%	Suggestive of monomeric prolactin
TSH	3.68 $\mu\text{IU/mL}$	0.45–4.5 $\mu\text{IU/mL}$

Hormonal profiles before and after amisulpride discontinuation are detailed in Table 2.

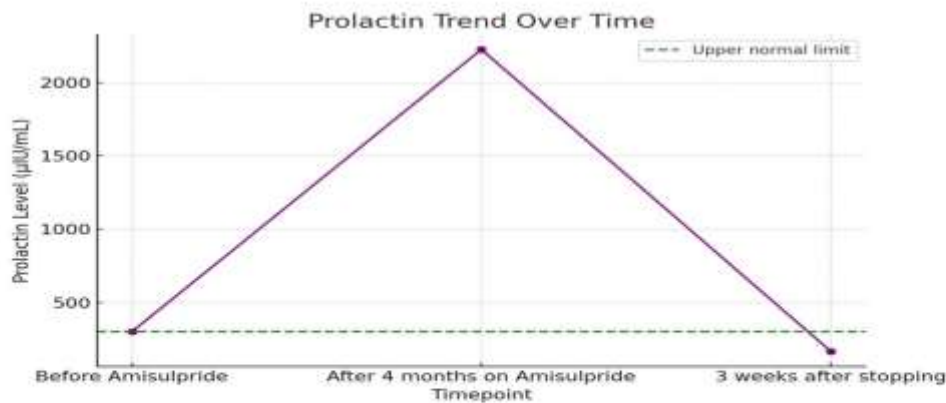


Figure 1: This graph shows the patient's prolactin levels before starting amisulpride, after 4 months on the medication, and 3 weeks after discontinuation.

Management and Follow-up

Amisulpride was withdrawn under psychiatric guidance. The patient was continued on olanzapine and venlafaxine, which had not previously caused any endocrine issues. She was monitored regularly, and gradual clinical improvement was noted. Her menstrual cycles resumed within 6–8 weeks of stopping amisulpride. Prolactin levels remained normal.

Discussion

Amisulpride is a therapeutic drug used in treating schizophrenia and depressive symptoms. Even though it is effective, it is associated with increased prolactin levels, even at low doses [4]. It operates in the tuberoinfundibular pathway in presynaptic and postsynaptic D2 receptors since it is primary in inhibiting prolactin secretion by dopamine[7,8]. In this case, the timing of the initiation of amisulpride and amenorrhea, as well as the subsequent symptom resolution, with drug discontinuation, confirmed the diagnosis of drug-induced hyperprolactinemia. Although they are not routinely used in psychiatry, prolactin assays should nonetheless be encouraged, especially in women of reproductive age who has menstrual disorders. In cases where antipsychotic treatment is unavoidable, alternatives like aripiprazole may be safer options [5][6].

Conclusion

This case emphasizes the need for awareness about the endocrine side effects of antipsychotics. Early recognition and appropriate therapy adjustment can reverse symptoms and reduce patient burden.

References

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