



**Clinical case**

**Levosulpiride-induced hyperprolactinemia: a case report from the endocrinology department of the Libreville University Hospital**

Hyperprolactinémie induite par lévosulpiride : à propos d'un cas au service d'endocrinologie du CHU de Libreville

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**Résumé**

Introduction : L'hyperprolactinémie est un trouble endocrinien aux étiologies variées. L'hyperprolactinémie d'origine médicamenteuse n'est pas rare. Nous rapportons le cas d'une patiente ayant développé une hyperprolactinémie après la prise de lévosulpiride.

Cas clinique : Mme L. B., âgée de 43 ans, a été adressée à notre consultation pour une galactorrhée évoluant depuis environ un mois. Deux mois auparavant, la patiente avait consulté un médecin pour des céphalées et des vertiges associés à des nausées et quelques épisodes de vomissements. Un traitement à base d'antalgiques de deuxième palier, d'un antivertigineux et d'une association prokinétique et antiémétique lui avait été prescrit. Environ six semaines après le début du traitement, la patiente a remarqué une tension mammaire, puis, à la pression, un écoulement lactescent, sans troubles du cycle menstruel. L'IRM hypophysaire était normale, mais le taux de prolactine était élevé à 40 ng/ml. Lors de notre consultation, la patiente a rapporté une

prise régulière de comprimés de lévosulpiride 25 mg (Dislep 25 mg), à raison d'un comprimé trois fois par jour pendant un mois. Le reste du bilan hypophysaire était sans particularité. La prise en charge a consisté à interrompre l'agent prokinétique. Deux semaines plus tard, une réduction de la sensibilité mammaire a été notée. Un mois après l'arrêt du médicament, son taux de prolactine sérique était de 29 ng/ml et ses symptômes dyspeptiques étaient bien contrôlés par un inhibiteur de la pompe à protons.

Conclusion : Compte tenu de l'utilisation croissante du lévosulpiride, une meilleure connaissance de son effet sur les taux de prolactine sérique semble nécessaire, notamment pour réduire les erreurs diagnostiques et optimiser les examens complémentaires.

Mots-clés : Hyperprolactinémie ; Lévosulpiride ; Effet indésirable ; Galactorrhée.

**Abstract**

Introduction: Hyperprolactinemia is an endocrine disorder with diverse etiologies. Drug-induced hyperprolactinemia is not uncommon. We report the

case of a patient who developed hyperprolactinemia after taking levosulpiride .

Clinical case: Mrs. L., B., aged 43, was referred to our clinic for galactorrhea that had been present for approximately one month. Two months prior , the patient had consulted a physician for headaches and vertigo associated with nausea and a few episodes of vomiting. The patient received treatment with second-line analgesics, an anti-vertigo medication, and a combination of prokinetics and an antiemetic. Approximately six weeks after the start of treatment , The patient noticed breast tenderness , followed by a milky discharge upon breast pressure, without any menstrual cycle irregularities. A pituitary MRI was normal, but her prolactin level was elevated at 40 ng /ml . During our consultation, the patient reported consistent use of levosulpiride 25 mg tablets ( Dislep 25 mg), one tablet three times a day for one month. The rest of the pituitary workup was unremarkable. Management consisted of discontinuing the prokinetic agent . Two weeks later, a reduction in breast tenderness was noted. One month after stopping the medication, her serum prolactin level was 29 ng / ml , and her dyspeptic symptoms were well controlled with a proton pump inhibitor.

Conclusion: Due to the increasing use of levosulpiride , greater awareness of its effect on serum prolactin levels appears necessary, particularly to reduce diagnostic errors and streamline further investigations.

Keywords: Hyperprolactinemia; Levosulpiride, Adverse effect, Galactorrhea.

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## Introduction

Hyperprolactinemia, characterized by a supraphysiological secretion of prolactin, is a common but underestimated symptom affecting approximately 1.5% of the general population. It can result from various causes, including treatment with certain medications. Levosulpiride is the levorotatory enantiomer of sulpiride, primarily used in the treatment of dyspeptic syndromes of various

origins. Its prokinetic effect is due to the blockade of type 2 (D2) enteric inhibitory dopamine receptors. However, antagonism of central D2 receptors induces both therapeutic effects (antiemetic effect related to D2 receptor blockade) and adverse effects (notably hyperprolactinemia ). We have described the case of a patient suffering from hyperprolactinemia following the use of levosulpiride . The objective here is to illustrate the precision required in the etiological approach to hyperprolactinemia, highlighting the crucial role of meticulous questioning in the diagnostic orientation.

## Clinical case

Mrs. L., B., a 43-year-old nulliparous woman with a history of allergic rhinitis, was referred to our clinic for galactorrhea that had been progressing for approximately one month . Two months prior , the patient had consulted a physician for headaches and vertigo associated with nausea and a few episodes of vomiting. Based on these symptoms , routine tests were ordered , the results of which were unremarkable. The patient received treatment with second-line analgesics, an anti-vertigo medication, and a combination of prokinetics and an antiemetic. Approximately six weeks after the start of treatment , The patient noticed breast tenderness , followed by a milky discharge upon breast pressure, without any menstrual cycle disturbances. In light of this new symptom, further investigations were ordered, including a pituitary Magnetic Resonance Imaging (MRI) scan with gadolinium contrast and a serum prolactin level measurement, which revealed a prolactin level of 40 ng / mL . The pituitary MRI was normal, as shown in Figures 1 and 2.

The patient was referred to a specialist center for further evaluation. During our consultation, the patient history revealed that she had been taking levosulpiride 25 mg tablets ( Dislep 25 mg) continuously, one tablet three times a day for one month. This treatment had been prescribed two months prior for her dyspeptic symptoms. The rest of the pituitary workup was

unremarkable. The results are summarized in Table I. Management consisted of discontinuing the prokinetic medication. Two weeks later, there was a regression of breast tenderness and one month after stopping treatment, the serum prolactin level was 29 ng / mL and dyspeptic symptoms were well controlled under proton pump inhibitor.

Table I: Initial hormonal assessment

Hormones	Measured rates	Laboratory standards
Prolactin	40 ng /ml	3-20ng/ml
TSHus	1.74 uIU/ml	0.27-4.7 uUI /ml
FSH	2.64 mIU/ml	3.03-8.08 mIU /ml
LH	4.31 mIU/ml	2-8 mIU /ml
FT3	3.97 pmol /l	4.00-8.30
FT4	9.96 pmol/l	9.00-25.00
Estradiol	231.65pg/ml	22-417 pg /ml

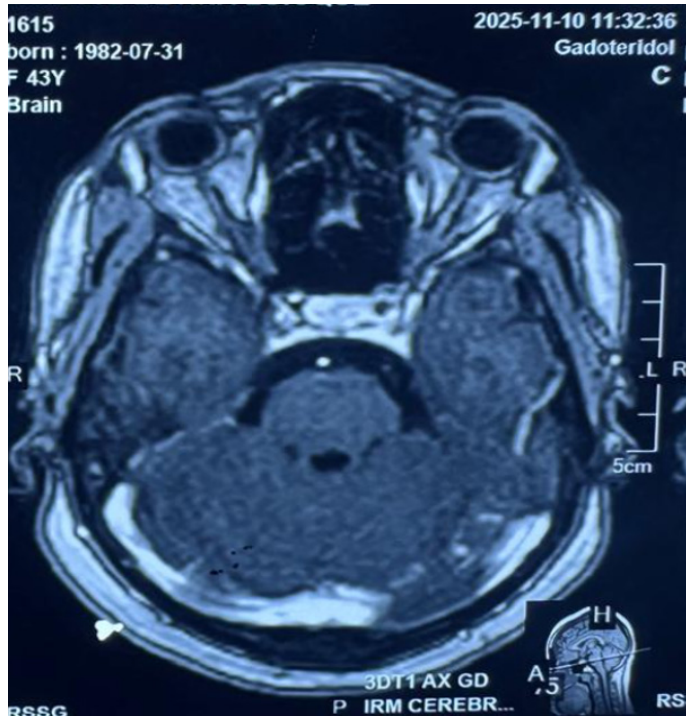


Figure 1: Pituitary gland seen in axial section on T1 sequence.

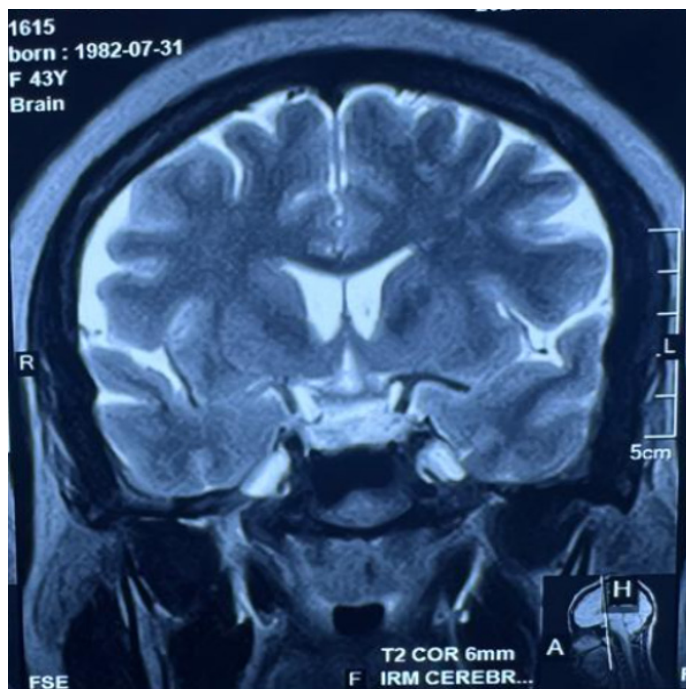


Figure 2: Pituitary gland seen in coronal section on T2 sequence.

### Discussion

This case describes significant hyperprolactinemia in a patient treated with levosulpiride , a dopamine antagonist frequently prescribed for digestive disorders. The limitation of this observation is that it represents a single case.

In this case, hyperprolactinemia was revealed by galactorrhea. Galactorrhea is a worrying symptom for the patient and unusual for general practitioners. Its etiology is often difficult to determine, leading to diagnostic investigations that are sometimes disproportionate and costly for patients. Hyperprolactinemia is a disorder of lactotropin secretion . Its etiologies are multiple: physiological situations such as breast manipulation and pregnancy; diseases (hypothyroidism and PCOS); certain medications; and pituitary disorders, primarily prolactinoma , are the causes (1) . In nearly half of cases, hyperprolactinemia is drug-induced; in approximately 25% of cases, it is linked to a pituitary microadenoma , and in 5% to early pregnancy (2) . Menstrual cycle irregularities and galactorrhea are the most frequent signs: more than 90% of women with hyperprolactinemia have amenorrhea or oligoamenorrhea . In our case, no menstrual irregularities were found, probably due to the relatively short time between the onset of hyperprolactinemia and diagnosis (3) . Galactorrhea was the symptom that alerted the patient; it can

remain unrecognized for a long time because breast self-examination or self-pressure is not a common practice among patients. Several therapeutic classes are known for their hyperprolactinemic effects . Indeed, antipsychotics are the drugs most frequently implicated in hyperprolactinemia (4) ; however, other classes of drugs that can induce hyperprolactinemia include antidepressants, antihypertensives, laxatives, antiemetics, and opioids (5). (6) Levosulpiride is the levorotatory enantiomer of sulpiride, used in the treatment of numerous conditions such as nausea and vomiting, dyspepsia, depression, and psychosis. Previously, prokinetic agents such as domperidone and metoclopramide have been reported to cause hyperprolactinemia . (7) . From the mid-1990s onwards, the elevation of prolactin under levosulpiride was described (8) . Lozano et al, in a prospective study of 342 participants, reported galactorrhea in 26.7% of patients taking levosulpiride (9) This result is consistent with the results we report in this case. It is explained by the prokinetic effect of levosulpiride , leading to blockade of enteric inhibitory D2 receptors (neuronal and muscular) and its interaction with type 4 serotonergic receptors (5-HT4). The serotonergic component of levosulpiride enhances its therapeutic efficacy in gastrointestinal disorders such as functional dyspepsia and diabetic gastroparesis (10) . Furthermore, antagonism of central D2 receptors can lead to both therapeutic (antiemetic due to D2 receptor blockade in CTZ) and adverse effects.

At the pituitary level, dopamine normally inhibits prolactin secretion. By blocking the D2 receptors of the pituitary gland, it suppresses the inhibitory effect of dopamine on lactotropin , resulting in hyperprolactinemia. Drug-induced hyperprolactinemia is often symptomatic but can also go unnoticed. It causes galactorrhea in 37.5% of women in a sample of 40 women with galactorrhea (11) . Prolactin levels may be below 100 ng / mL in patients with microprolactinomas, pseudoprolactinomas, drug-induced hyperprolactinemia, or systemic diseases (12) . A medical history should always be obtained from all patients with galactorrhea or hyperprolactinemia

before considering further evaluation. Treatment primarily involves discontinuing the offending drug and switching to another drug that does not induce hyperprolactinemia, using hormone replacement therapy (estrogen or testosterone), or, more rarely, the cautious addition of a dopamine agonist. Normalization of levels is observed within days to weeks of stopping treatment (13) . The use of dopamine agonists is not routine. We observed a regression of symptoms and prolactin levels after a few weeks of treatment discontinuation in the present case, as described in the literature. This case therefore underscores the importance of systematically considering a drug-induced cause in all cases of hyperprolactinemia, in order to avoid unnecessary and costly investigations. Early recognition of an iatrogenic etiology allows for rapid guidance of management, and identifying drug-induced hyperprolactinemia can prevent disproportionate further investigations.

## Conclusion

In cases of hyperprolactinemia, a thorough patient history is essential to identify any exposure to substances that may increase prolactin secretion. Given the increasing use of levosulpiride , greater awareness of its effect on serum prolactin levels is necessary, particularly to reduce diagnostic errors and streamline further investigations. Therefore, the appropriateness of prescribing levosulpiride should be assessed , and the duration of treatment should be limited with close clinical monitoring. Discontinuation of treatment is the first diagnostic and therapeutic step.

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**Conflict of interest :** None

## References

- [1] Pralong FP, Gaillard RC. Hyperprolactinemias [Internet]. [cited 2025 Dec 15]. Available from: [https://www.gfmer.ch/Presentations\\_Fr/Hyperprolactinemies.htm](https://www.gfmer.ch/Presentations_Fr/Hyperprolactinemies.htm)
- [2] Vialle Laboratory. Hyperprolactinemia and diagnostic approach [Internet]. [cited 2025 Dec 15]. Available from: <https://www.labovialle.com/archives/333-articles-parus-en-2023/887-hyperprolactinemie-et-demarche-diagnostique>
- [3] French Society of Endocrinology. Pituitary adenoma [Internet]. [cited 2025 Dec 15]. Available from: <https://www.sfendocrino.org/item-244-adenome-hypophysaire/>
- [4] Maiter D. Moderate hyperprolactinemia in general practice [Internet]. [cited 2025 Dec 15]. Available from: [https://www.ssmg.be/wp-content/uploads/RMG/407/RMG407\\_06-13.pdf](https://www.ssmg.be/wp-content/uploads/RMG/407/RMG407_06-13.pdf)
- [5] Molitch ME. Medication-induced hyperprolactinemia. *Mayo Clin Proc.* 2005;80(8):1050-1057.
- [6] Bonnier de La Chapelle M. Hyperprolactinemia: from diagnosis to treatment according to etiology [Internet]. [cited 2025 Dec 7]. Available from: [https://www.realites-cardiologiques.com/wp-content/uploads/sites/6/2024/03/03\\_BONNIER\\_RGO.pdf](https://www.realites-cardiologiques.com/wp-content/uploads/sites/6/2024/03/03_BONNIER_RGO.pdf)
- [7] Healy DL, Burger HG. Sustained elevation of serum prolactin by metoclopramide: a clinical model of idiopathic hyperprolactinemia. *J Clin Endocrinol Metab.* 1978;46(5):709-714.

- [8] Mucci G, Nolfi M, Maj M. Levosulpiride: a review of its clinical use in psychiatry. *Pharmacol Res.* 1995;31(2).
- [9] Lozano R, Concha MP, Montealegre A, de Leon L, Villalba JO, Esteban HL, et al. Effectiveness and safety of levosulpiride in the treatment of dysmotility-like functional dyspepsia. *Ther Clin Risk Manag.* 2007;3(1):149-155.
- [10] Kuchay MS, Mithal A. Levosulpiride and serum prolactin levels. *Indian J Endocrinol Metab.* 2017;21(2):355-358.
- [11] Atluri S, Sarathi V, Goel A, Boppana R, Shivaprasad C. Etiological profile of galactorrhoea. *Indian J Endocrinol Metab.* 2018;22(4):489-493.
- [12] Vilar L, Fleseriu M, Bronstein MD. Challenges and pitfalls in the diagnosis of hyperprolactinemia. *Arq Bras Endocrinol Metabol.* 2014;58(1):9-22.
- [13] Levosulpiride-induced hyperprolactinemia. *J Coll Physicians Surg Pak.* 2020;30(4):457.

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