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Bilateral Galactorrhea Revealing Idiopathic or Non-Tumoral Hyperprolactinemia: A Case Report and Review of the Literature

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Abstract **Case Report**

Prolactin is a polypeptide hormone that is synthesized in the anterior pituitary gland and secreted in a pulsatile manner. It plays central role in a variety of reproductive functions and lactation. Prolactin release in humans depends on physiological state and varies in response to different stimuli. Hyperprolactinemia is a common endocrinological disorder; it could be physiological, pathological or idiopathic in origin. In several respects prolactin is unique among anterior pituitary hormones. We report the case of a 25-year-old woman who came to the endocrinology department with galactorrhea. The history revealed spontaneous and provoked galactorrhea, with secondary amenorrhea that had been going on for 05 years and had been neglected by the patient. The questioning did not reveal any particular medication being taken. Our examination revealed induced lactescent galactorrhea and moderate hirsutism with a Ferriman score of 18. Pregnancy was ruled out, Prolactin=148.1 ng/mL (6xN), the rest of the hypophysiogram was unremarkable (no associated hypogonadism). A pituitary magnetic resonance imaging (MRI) revealed no pituitary adenoma or interruption of the pituitary stalk! We adopted the diagnosis of symptomatic non-tumor hyperprolactinemia. Cabergoline was introduced at 0.5 mg per week. The course of the disease was favorable, with a reduction in galactorrhea and normalization of the menstrual cycle.

Keywords: Hyperprolactinemia, Prolactin, Pituitary, Cabergoline, Galactorrhea, Amenorrhea.

INTRODUCTION

Prolactin is a peptide hormone synthesized by the lactotrophic cells of the pituitary gland. Its physiological action is limited to lactation in humans, but its role in pathology is important. Between 1% and 1.5% of women are thought to have hyperprolactinemia; 20% of women with hypothalamic-pituitary amenorrhea and 75% of those with amenorrhea and galactorrhea have hyperprolactinemia [1, 2]. Hyperprolactinemia causes gonadal dysfunction by interfering with the secretion of GnRH (Gonadotropin releasing hormone), which inhibits the release of the pituitary gonadotropins LH and FSH [3]. When it is linked to an adenomatous pathology, it is most often associated with a microprolactinoma (< 10 mm, 90% of cases), more rarely with a macroprolactinoma.Hyperprolactinemia syndromes are varied, most often not serious, and the question of the choice of treatment or even isolated monitoring frequently arises.

CASE REPORT

A 25-year-old woman presented with galactorrhea. The history revealed spontaneous and provoked galactorrhea, with secondary amenorrhea that had been going on for 05 years and had been neglected by the patient. The questioning did not reveal any particular medication being taken. Our examination revealed induced lactescent galactorrhea and moderate hirsutism with a Ferriman score of 18. Pregnancy was ruled out, Prolactin=148.1 ng/mL (6xN), the rest of the hypophysiogram was unremarkable (no associated hypogonadism). A pituitary MRI revealed no pituitary adenoma or interruption of the pituitary stalk! We adopted the diagnosis of symptomatic non-tumor hyperprolactinemia. Cabergoline was introduced at 0.5 mg per week. The course of the disease was favorable, with a reduction in galactorrhea and normalization of the menstrual cycle.

DISCUSSION

The clinical manifestations are generally related to the influence of prolactin on the gonadotropic axis, more rarely to the risks associated with the tumor volume of the adenoma. In genitally active women, hyperprolactinemia is most often discovered in the setting of secondary amenorrhea or infertility [3]. Galactorrhea is present in 80% of cases. Some women with hyperprolactinemia also have spaniomenorrhea, or even regular cycles [3]. In most cases, hyperprolactinemia is related to a microadenoma, with no impact on the visual field, as in our patient's case. Diagnosis is based on prolactin measurement, which should be requested for each of the clinical signs mentioned. If the hyperprolactinemia is moderate (< 50 ng/mL), prolactin assays should be repeated, or even a search made for the presence of big-big prolactin in the absence of a suggestive clinical syndrome (probably the result of a link between glycosylated prolactin and immunoglobulins, which can give rise to "false" hyperprolactinemia) [4, 5]. It is important to ensure that no drugs are being taken that interfere with the prolactin assay, and that the patient is not pregnant, hypothyroid or suffering from renal failure**.** In prolactinomas, there is a good correlation between prolactin levels and tumor size. In the case of hyperprolactinemia exceeding 200 ng/mL, the diagnosis of a macroprolactinoma is almost certain [6]. In the case of a prolactin level of between 100 and 200 ng/mL, as in our patient's case, the diagnosis may point either to a tumor origin (micro or macroadenoma) or to hyperprolactinemia due to disconnection, and rarely to a drug-related cause [6]. In the absence of a cause, hyperprolactinemia is idiopathic or non-tumoral. It may be related to a microprolactinoma, which is too small to be detected on MRI. It is managed in the same way as a microadenoma.

CONCLUSION

The decision to treat hyperprolactinemia is based on the size of the tumor and its impact on gonadotropic function. The first-line treatment of prolactin adenomas is based on the use of dopaminergic agonists (bromocriptine, quinagolide, cabergoline), due to their very good efficacy and the risk of recurrence after surgery [7]. Establishing the aetiological diagnoses of hyperprolactinemia is a crucial step in its management.

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