

# HYPERPROLACTINEMIA ASSOCIATED TO CALCIFICATION OF THE PITUITARY STALK

## CASE REPORT

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**ABSTRACT** - In this work, the authors report the case of a female patient with 24 years of age with hyperprolactinemia, who presented a pituitary stalk calcification as seen by CT scan. Once other possible etiologies were excluded, we concluded that the calcification was probably related to hyperprolactinemia caused by interruption of the input of dopamine to the pituitary gland.

**KEY WORDS:** pituitary gland, prolactin, hyperprolactinemia, pituitary stalk, calcification.

### **Hiperprolactinemia associada a calcificação da haste hipofisária: relato de caso**

**RESUMO** - Os autores relatam o caso de uma paciente feminina, de 24 anos, com hiperprolactinemia, na qual a tomografia computadorizada de sela túrcica detectou uma calcificação na haste hipofisária. Uma vez excluídas outras etiologias possíveis, concluiu-se pela provável relação causal entre a presença da calcificação e a hiperprolactinemia, em consequência da interrupção do aporte dopaminérgico à glândula pituitária.

**PALAVRAS-CHAVE:** hipófise, prolactina, hiperprolactinemia, haste hipofisária, calcificação.

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The sustained hyperprolactinemia can result from the use of medication, neurogenic disorders, hyperthyroidism, chronic renal failure, cirrhosis and hypothalamic or pituitary diseases of which prolactinomas are highlighted<sup>1</sup>. Among the alterations of the pituitary stalk which are related to hyperprolactinemia are its deviation from the median line, eventually suggesting the presence of pituitary adenoma and secondary compression of a pseudoprolactinoma<sup>2</sup>.

The aim of this study is to report the case of a patient with hyperprolactinemia, probably due to the interruption of the pituitary stalk by calcification. This situation, as far as we know, has not been reported in the literature.

## CASE REPORT

Female patient, white, 24 years of age, presented with amenorrhea for two years. She was using 2.5 mg per day of bromocriptine for 5 months, which was prescribed after the repeated observation of hyperprolactinemia of 200 ng/ml (normal reference range=3-24) and estradiol of 17 pg/ml (30-150). The patient referred decreasing of libido and denied other kind of complaints. She reported seizure since 13 year of age, with last episode at 12 months ago, when she was under treatment with phenobarbital. At physical examination, she weighted 61.2 kg,

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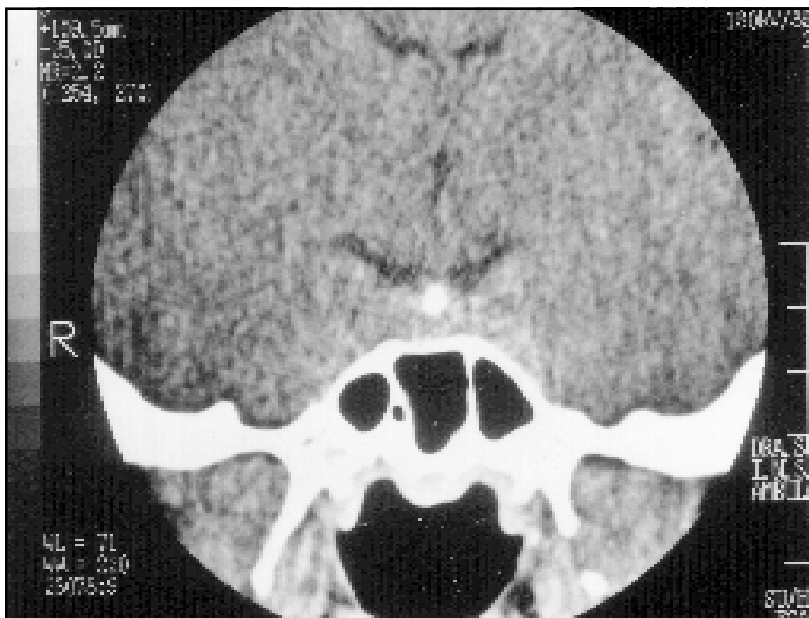


Fig 1. CT scan of the sellar region showing calcification of the pituitary stalk.

presented with galactorrhea and a small diffuse goiter. The prolactin concentration at that time was 116 ng/ml, the functional tests of thyroid were normal and CT scan was negative for sellar lesion, detecting calcification of the pituitary stalk (Fig 1).

From that moment onwards, the dosage of bromocriptine was progressively increased. Her period was re-established and there was an improvement of the libido with 5 mg/day, but with persistency of hyperprolactinemia. The minor concentration in serum concentration of prolactin was 29 ng/ml with the use of 12.5 mg of bromocriptine.

## DISCUSSION

The hyperprolactinemia presented in this case is not related to the use of drugs or to hypothyroidism. Nevertheless, the presence of microprolactinoma cannot be ruled out, since CT scan of the sellar region, which is useful in the definition of bone alterations, not always presents enough sensibility for the diagnosis of microadenomas<sup>3</sup>. The most probable etiology of the hyperprolactinemia observed here is a disturb in the neuroendocrine mechanisms which control prolactin secretion, represented by the interruption of the input of dopamine to the pituitary gland. High levels of prolactin, higher than 150 ng/ml, which are classically suggestive of a tumoral etiology, should not be used alone as a discriminating factor for the presence or absence of an adenoma which was not identified through imaging methods. In a recent series of pseudoprolactinoma, with histological confirmation of the absence of immunohistochemical reactivity for prolactin in all tumors, 7% of the patients presented serum concentration of prolactin higher than 200 ng/ml, reaching a value of 504 ng/ml in one case<sup>4</sup>.

The pituitary stalk, with origin in the median eminence and insertion in the neurohypophysis, measures approximately 3.2 mm of transverse diameter in the level of the optic chiasma and 1.9 mm in the level of the pituitary insertion<sup>5</sup> and can present anatomic and/or functional alterations. The stalk is anatomically absent in cases of typical congenital hypopituitarism with diabetes insipidus<sup>6</sup>, and as a consequence of previous trauma<sup>7</sup>. It can be also found deviated from the medium line, being

this inclination previously interpreted as suggestive of the presence of pituitary adenoma. This concept has been going through modifications, since it has been noticed that the inclination of the stalk can occur in up to 46% of the general population, even by eccentricity of the pituitary gland in relation to the median line of the brain, or by an eccentric insertion of the pituitary infundibulum in relation to the gland median line, representing a variation of normality<sup>8</sup>. A third anatomical alteration is the thickening of the stalk associated to lymphocytic pituitary-infundibulum<sup>9</sup>, granulomatous diseases<sup>10</sup> or local or metastatic tumors<sup>11</sup>.

Finally, from the functional point of view, the stalk can suffer compression by a tumoral mass which obstructs the capillaries, blocking the input of hypothalamic dopamine (compression syndrome or stalk section effect). These tumors, designated as pseudoprolactinomas, are usually non-functional pituitary adenomas, but also craniopharyngiomas and other parasellar tumors. As far as we know, there are no previous reports in the literature on calcification of the pituitary stalk, which would behave as a space-occupying lesion, interfering with the transit of neurotransmitters in this via.

Pathologic calcifications in the central nervous system, when non idiopathic, are related to various causes, among them metabolic disturbs, specially of calcium and phosphorus, toxic-anoxic, vascular, and tumoral causes, and parasitic and infectious diseases. Independently of its cause, the calcification process is similar in relation to the structure and chemical composition. In the case presented here, it is difficult to establish the diagnosis of the primary event which originated the calcification, due to the absence of data about the updated and previous history or findings on physical examination suggestive of a systemic disease. At the same time, there are no tomographic alterations in the central nervous system, as well as thickening of the stalk compatible with inflammatory lesion or diagnosis of diabetes insipidus. The magnetic resonance imaging, unaccessible to the patient in the financial point of view, is the gold standard method for the evaluation of the sellar and parasellar region<sup>12</sup>. However the possible detection of a microadenoma would not definitely clarify the etiology of the hyperprolactinemia, as the anatomic lesion of the stalk would persist. On the other hand, the finding of minimal lesions suggesting granulomatous disease could add more information about the etiology of the calcification.

In conclusion, the authors describe another lesion of the central nervous system related to hyperprolactinemia, represented by the presence of calcification of the pituitary stalk, which compromises the input of dopamine from the nervous terminals of the median eminence.

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