

Case Report**“Macro-adenoma or Pituitary Hyperplasia”: How to Make a Difference and don’t Operate? About Case Report****Sanae Elhadri, Faycal Elguendouz, Hicham Baïzri***

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Abstract: Pituitary hyperplasia is non-neoplastic growth of at least one line of pituitary cells. The thyroid stimulating line can be affected with the waning of a device drawing hypothyroidism. It is rare and under diagnosed. Its main differential diagnosis is pituitary adenoma. We report the case of a 22 old-year patient, addressed for etiological and presurgical evaluation of a macro-adenoma. The history found a spaniomenorrhoea since puberty and galactorrhea since 6 months without endocrine syndrome or endocranial tumor syndrome. Hormonal measures objective a moderate hyperprolactinemia and TSH > 100 IU / ml with a low LT4 3.24 pmol / l. Pituitary MRI reveal a pituitary tumor process engaging the optic chiasm without compressing it. The patient is put under L-Thyroxine with normalization of thyroid status and prolactin levels and a very significant regression of pituitary hyperplasia after one year of treatment. Although classical, pituitary hyperplasia secondary to hypothyroidism is rare and seldom reaches such a large size. It results from the overproduction of TRH by loss of the negative feedback exerted by thyroid hormones. The knowledge of this disease entity is paramount and should allow the clinician to distinguish an authentic tumor process to prevent the patient unnecessary surgery, heavy and not without risk.**Keywords:** Primary hypothyroidism, Macro-adenoma, Pituitary hyperplasia.

INTRODUCTION

Pituitary hyperplasia secondary to primary hypothyroidism has been well documented in the literature [1]. It results from the loss of negative feedback of thyroxine on thyrotroph axis and subsequent overproduction of TRH (thyrotroph releasing hormone) which causes both enlarged pituitary of two lines cells: lactotroph and thyrotroph [2, 3]. Despite recent advances in imaging techniques, the distinction between authentic macro-adenoma and hyperplasia of thyrotropic pituitary cells can be difficult even on an MRI.

We report the case of a 22 old-year young woman who was going to be operated for a macro-adenoma. Hormonal measures and good analysis of pituitary MRI was in favor of pituitary hyperplasia related a deep and unrecognized hypothyroidism.

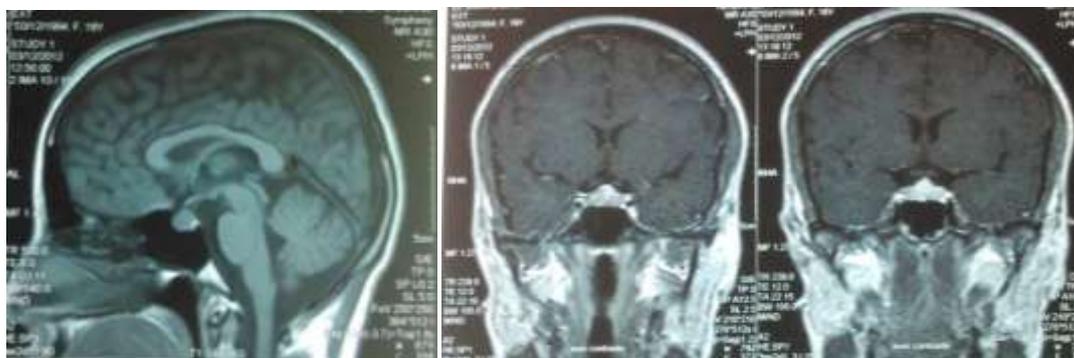
CASE REPORT

A 22 old-year young woman, with no particular medical history, menarche at age of 13 years. A provisional diagnosis of pituitary macroadenoma was made and the patient was referred for an endocrinology

consultation before a planned surgical adenomectomy. The history found a spaniomenorrhoea since puberty and galactorrhea since 6 months ago without endocrine syndrome nor endocranial tumor syndrome. Laboratory investigations revealed moderate hyperprolactinemia at 85 ng / l (VN: 4.1 -29 ng / l), grossly increased TSH > 100 IU / ml (VN: 0.27 -4. 20 IU / ml) with a low LT4 3.24 pmol / l (VN: 11.5 to 22.7 pmol / l), low 17 β-estradiol at 42.60 pg / ml (VN: -211 43.8 pg / ml), low level of LH <0.2 IU / l (VN: 1 11.4 IU / l), FSH at 2.10 IU / l (VN: 1.7 to 7.7 IU / l), 8 a.m. cortisol , urinary free cortisol/day and IGF1 were normal. Pituitary MRI revealed a pituitary tumor process (10mm), rounded, although limited, elevating the sellar diaphragm and contacting the optic chiasm without compressing it (Fig. 1). The visual field examination showed two notches: at the lower right nasal region and at the lower left temporal region. The patient has been under L-Thyroxine with a return of menstruation, the disappearance of galactorrhea, normalization of thyroid status and prolactin levels after two months of treatment (Table 1) and a very important regression of pituitary hyperplasia after one year of treatment (Fig. 2).

Table 1: Results of laboratory evaluations

| Parameter | Before treatment | After treatment | Normal Values |
|-----------|------------------|-----------------|--------------------|
| TSH | > 100 UI/ml | 1.6 UI/ml | 0.27 – 4.20 UI/ml |
| L-T4 | 3.24 pmol/l | 16.7 pmol/l | 11.5 – 22.7 pmol/l |
| Prolactin | 85 ng/l | 20.5 ng/ml | 4.1 -29 ng/ml |

**Fig. 1: Pituitary MRI before treatment with L-Thyroxine****Fig. 2: Pituitary MRI one year after treatment with L-Thyroxine**

DISCUSSION

In 1851, Niépce was the first to raise the possibility of increasing the volume of the pituitary gland in primary hypothyroidism [4]. The incidence of pituitary hyperplasia in patients with hypothyroidism varies between 25% and 81% [5]. Khawaja *et al.* reported the presence of the pituitary hyperplasia in 70% of patients with TSH levels ≥ 50 $\mu\text{IU} / \text{ml}$ and a statistically significant positive correlation between the size of the volume of the pituitary gland and the levels of TSH [6]. Normally, circulating thyroid hormones act by a negative feedback on the TRH secretion. If the thyroid secretion is insufficient, the level of serum TRH increases, finally causing thyrotropic hyperplasia cells and subsequent enlargement of the pituitary [2]. TRH has also a small effect on stimulating lactotroph cells explaining the hyperprolactinemia found in about 3/4 of cases [5]. This overproduction of prolactin may be secondary to the decrease in the dopamine secretion by compression of the pituitary stalk [7]. Histological examination of the pituitary gland in some patients with primary hypothyroidism showed hyperplasia of lactotroph and thyrotropic lines compared with normal gland [8]. Probably our patient was not a significant component of lactotroph hyperplasia. The slight increase in prolactin was probably due to the mass compression of the infundibulum.

Pituitary hyperplasia is a physiological response that can occur at birth, at puberty, during pregnancy and after childbirth. During puberty, the pituitary height can reach 8 mm for males and 10 mm for females. During pregnancy the gland grows up to 10 mm in height, and immediately after delivery, it can reach 12 mm high. After the first week postpartum, the gland quickly returns to normal [9].

In imaging, very important and symmetrical pituitary hyperplasia can mimic a pituitary adenoma or hypophysitis [9]. The interpretation of a pituitary mass without endocrine investigation may lead to unnecessary surgery with potentially catastrophic results [7, 10-12]. Analysis of some subtle signs MRI allows for the differential diagnosis between hyperplasia and pituitary macroadenomas. First, pituitary hyperplasia is generally homogenous appearance after administration of gadolinium on T1 sequences. A pituitary macro-adenoma can be either homogeneous or heterogeneous, but whose size exceeds 10 mm with deviation of the pituitary stalk [13, 14]. Then, when the pituitary enlargement is median with regular contours, it is a sign for pituitary hyperplasia [1]. Finally, the signal intensity of the physiological posterior pituitary can be absent in 20% of patients

having a macro-adenoma but present in those with hyperplasia [13].

Replacement therapy with thyroid hormones leads to resolution of symptoms and regression of suprasellar mass as was the case in our patient. Khawaja *et al.* reported that in 85% of patients with pituitary enlargement and who underwent follow-up examinations by MRI, there's been a reduction in the gland size after treatment with L-thyroxine [6].

CONCLUSION

Our case illustrates a reactional pituitary tumor syndrome with peripheral hypothyroidism. Although classical, pituitary hyperplasia due to hypothyroidism is rare and seldom reaches such a large size. Several similar cases have been reported with different radiological aspects. Knowledge of the various aspects of pituitary hyperplasia is paramount and should allow the clinician to make the difference with a veritable tumor process and to prevent the patient from unnecessary, heavy and not without risk surgery.

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