Turkish Journal of Endocrinology and Metabolism, (2004) 2 : 69-70 **CASE REPORT**

Primary Hypothyroidism and Hyperplasia of Thyrotrophic Cells

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We present a case, including history, laboratory findings and results of diagnostic imaging during a two year follow up period. A review of the pertinent literature is provided. A 40 year-old woman was referred to a neurologist for evaluation of frontal headache. Magnetic resonance imaging of the sella revealed a pituitary tumor and the patient was referred to a neurosurgeon for work up and surgery. The patient ended up in endocrinology when she became aware that the pituitary gland is an endocrine organ. The biochemical IU/l, FT3 1.91μevaluation of the anterior pituitary revealed a TSH of 243.8 pg/ml, FT4 0.45 ng/dl and anti-TPO >1000 IU/ml, the rest being within the normal range. Levothyroxine replacement therapy releaved the patient of her pertinent symptoms and caused shrinkage of pituitary hyperplasia. This case illustrate that primary hypothyroidism may be associated with pituitary enlargement. The primary care physician should consult an endocrinologist for complete evaluation in order to avoid unnecessary medical work up and surgical intervention

Key words: Hashimoto thyroiditis, headache, pituitary hyperplasia

Introduction

Pituitary enlargement secondary to primary hypothyroidism is a known but uncommon occurrence, which can be difficult to distinguish on computerized tomography (CT) and magnetic resonance imaging (MRI) from primary pituitary tumors. We describe a 40 year old female who was referred to a neurosurgeon for removal of a pituitary mass. The markedly elevated thyrotrophin stimulating hormone (TSH), absence of clinical features of hyperthyroidism, and low thyroid hormone values led to a diagnosis of pituitary enlargement secondary to primary hypothyroidism. The pituitary gland returned to normal size with thyroxine replacement therapy.

Case Report

A 40 year old female patient visited the endocrinology clinic as self referral stating that before the neurosurgeon takes out her pituitary gland she has decided to seek a second opinion. She had been complaining about a frontal headache for one week before she saw a neurologist who ordered a cranial MRI that revealed the pituitary tumor (Fig. 1). Her medical history was uneventful and findings on physical exam were unremarkable except for a palpable diffuse thyroid gland. On evaluation of the function of the anterior hypo-physis; estradiol 57.0 pg/ml (n.v. 23-361 pg/ml), FSH 2.1 mIU/ml (n.v.3.5-12.5 mIU/ml), LH 2.2 mIU/ml (n.v. 1.8-11.4 mIU/ml), 8 a.m. serum cortisol 12.1 μg/dl (n.v. 5-28 μg/dl), and ACTH < 10 pg/ml (n.v. 0-46 pg/ml), GH 0.5 ng/ml (n.v. 2-6 ng/ml) and IGF-I 156 μg/l (n.v. 78-258 μg/l), prolactin 19.2 ng/ml (n.v. 3.4-24.1 ng/ml), FT3 1.91 pmol/l (n.v. 3-9 pmol/l), FT4 0.45 ng/dl (n.v. 0.93-1.77 ng/dl), TSH 243.8 μIU/l (n.v. 0.27-4.2 μIU/l), and anti-TPO >1000 IU/ml (n.v. < 100 IU/ml). She was started on levothyroxine that was titrated up to 125 μg/day.

At the end of the sixth month of replacement therapy her FT4 was 1.04 ng/dl and TSH 0.9 μIU/l. Control MRI revealed a regression of the pituitary enlargement and disappearence of the tumor (Fig. 2).

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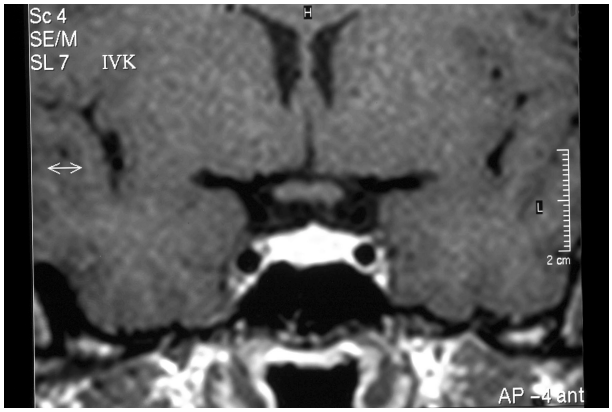

Figure 1. MRI image of the pituitary gland in hypothyroid state. The gland volume is enlarged in both size.

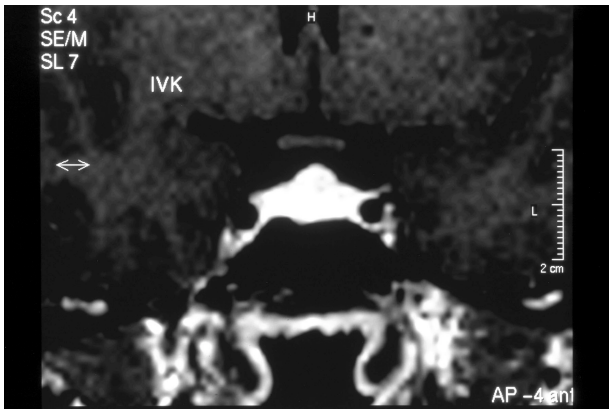

Figure 2. MRI image of the pituitary gland in eythyroid state. The gland volume is remarkably decreased in fig. 2 compared with fig. 1.

Discussion

Circulating thyroid hormones normally act by means of negative feedback on the hypotalamic secretion of thyrotropin-releasing hormone (TRH). If the thyroid gland secrets an insufficient quantity of thyroid hormones, the serum levels of TRH will increase (1). This eventually will result in hyperplasia of thyrotropin- producing cells (thyrotrops) and in subsequent enlargement of the pituitary gland (2). Thyrotrops are commonly located in the anteromedial portion of the anterior lobe and make up about 15% of the adenohypophysial cells (3). Because TRH has a weak stimulatory effect on lactotrophs hyperprolactinemia may also occur (4,5). In our case, serum prolactin level was normal. Histologic examinaton of the pituitary gland in some patients with primary hypothyroidism has shown hyperplasia of both thyrotrops and lactotrophs in an otherwise normal gland (5).

With the advent of Computed Tomography and MR imaging, directly visualization of the pituitary gland provided noninvasive volumetric confirmation of pituitary enlargement in patients with hypothyroidism and subsequent regression after replacement therapy with thyroid hormones (4).

In conclusion, the rapid progression of hyperplasia of pituitary gland following hypothyroidism and a regression of this hyperplasia probably due to thyroid replacement therapy is demonstrated by using MR volumetric technique.

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