

CLINICAL IMAGE

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A 26-year-old man with proptosis and a pituitary mass

Dennis Delasi Nyanyo, Amy Wheeler, Lisa B Nachtigall

CASE REPORT

A 26-year-old man presented for a routine annual physical. He endorsed weight gain with an otherwise negative review of systems. His vital signs were normal, and his exam was notable for well-proportioned obesity with mild proptosis. Initial thyroid function tests revealed a serum thyroid stimulating hormone (TSH) level of 2082 $\mu\text{U/mL}$ (0.40–5.00) and a serum T4 level of <0.4 ng/dL (0.9–1.8). Follow-up biochemical testing revealed an elevated prolactin, low testosterone (with suppressed LH and inappropriately normal FSH), and low insulin-like growth factor 1 (IGF-1) levels (Table 1). A brain magnetic resonance imaging (MRI) was obtained that showed a pituitary mass with suprasellar extension (Figure 1). Thyroid hormone was initiated. Thyroid replacement was associated with a decrease of his TSH, but his TSH levels remained slightly elevated intermittently during the initial phase of therapy of levothyroxine when he was non-compliant. His dose was uptitrated to 200 μg per day and on this dose of thyroid hormone with increased compliance, his TSH level normalized at 0.97 $\mu\text{U/mL}$, 18 months after beginning thyroid hormone therapy. Clinical evaluation at this point was remarkable only for persistent mild proptosis. Biochemical evaluation at approximately 18 months showed spontaneous normalization of IGF-1, testosterone, and prolactin. An interval brain MRI done at six months after the initial thyroid hormone replacement when his TSH was in the 15 to 20 range revealed a decreased size in the pituitary mass. Subsequently, a brain MRI obtained three years

after initiating thyroid hormone replacement, and 18 months after consistent normalization of TSH, showed complete regression of the pituitary mass (Figure 1).

Table 1: Hormonal test results at diagnosis

Hormone test	Result	Ref range	Units
Thyroxine	<1.0	(4.5–10.9)	ug/dL
Free T4	<0.4	(0.9–1.8)	ng/dL
T1-TSH	2082	(0.40–5.00)	uU/mL
Prolactin	52.3	(0.0–15.0)	ng/mL
Thyroid autoantibodies	661.1	(<0.3)	U/mL
Testosterone	123	(270–1070)	ng/dL
IGF-1	69	(114–492)	ng/mL

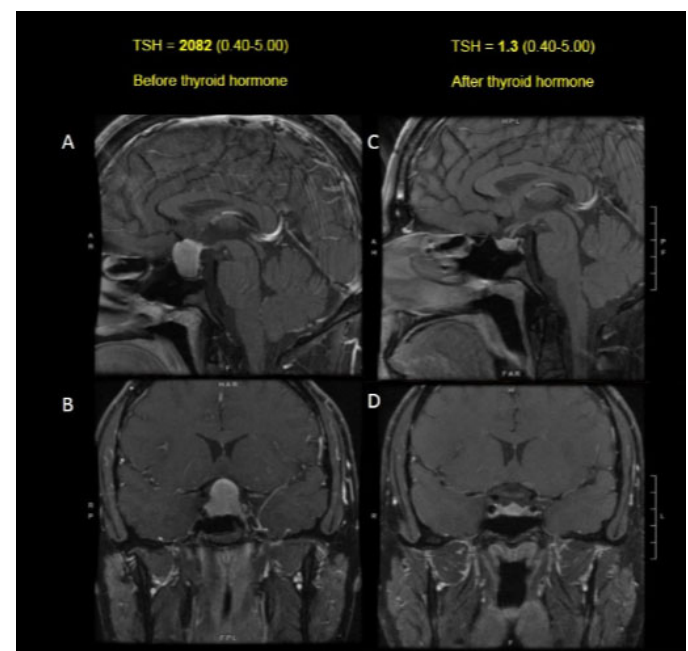


Figure 1: Pre- and post-treatment pituitary MRI. (A) Pre-treatment sagittal T1 post-contrast MRI of the brain and pituitary. (B) Pre-treatment coronal T1 post-contrast MRI of the brain and pituitary. (C) Post-treatment sagittal T1 post-contrast MRI of the brain and pituitary. (D) Post-treatment coronal T1 post-contrast MRI of the brain and pituitary.

Dennis Delasi Nyanyo¹, Amy Wheeler², Lisa B Nachtigall³

Affiliations: ¹Medical Student, Harvard Medical, Boston, MA, USA; ²MD, Adult Medicine Unit Chief, Revere Healthcare Clinic, Massachusetts General Hospital, Boston, MA, USA; ³MD, Clinical Director, Neuroendocrine & Pituitary Tumor Clinical Center, Massachusetts General Hospital, Boston, MA, USA.

Corresponding Author: Lisa B Nachtigall, MD, Neuroendocrine & Pituitary Tumor Clinical Center, Massachusetts General Hospital, 100 Blossom Street, Cox 140, Boston, MA 02114, USA; Email: Lnachtigall@mgh.harvard.edu

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DISCUSSION

Pituitary hyperplasia, defined as the enlargement of the pituitary gland with changes in pituitary gland structure, can occur as a result of primary hypothyroidism. Primary hypothyroidism is most commonly caused by autoimmune thyroiditis or Hashimoto's thyroiditis in the United States and by iodine deficiency worldwide [1]. It is characterized by a low T₄ and an elevated TSH [1].

The lack of thyroid hormone production by the thyroid leads to a loss of negative feedback on the hypothalamus and pituitary glands which results in an increase in production of thyrotropin-releasing hormone (TRH) which causes hyperplasia of thyrotropes in the pituitary and consequently increases the production of thyroid stimulating hormone [2]. Thyrotropin-releasing hormone has a stimulatory effect on lactotrophs which may result in hyperprolactinemia as was noted in our patient.

In addition to the typical signs of hypothyroidism such as cold intolerance, weight gain, constipation, and bradycardia, patients may also present with headache, vision loss, or other neurologic disturbances due to mass effect on the gland or surrounding tissue in the cavernous sinus as well as other endocrine disturbances. Women may present with symptoms such as amenorrhea and galactorrhea due to an elevated prolactin. When present in children, pituitary hyperplasia has also been associated with growth retardation [3].

While the cause of proptosis is not totally clear in this case, the presence of thyroid stimulating autoantibodies has been shown to be associated with thyroid associated ophthalmopathy and most likely explains the mild proptosis noted [4]. Although orbitopathy is more common in Graves' disease, there are cases of orbitopathy reported in Hashimoto's thyroiditis as well [4]. It is appropriate to evaluate pituitary function in patients with pituitary masses. In this case, the low IGF-1 and low testosterone were due to suppression of the hypothalamic-growth hormone axis and hypothalamic-pituitary-gonadotropic axes respectively, which normalized with resolution of the lesion.

It is important to differentiate pituitary hyperplasia from a pituitary thyrotroph adenoma although it is difficult to do this based on radiographic features alone. As a result, it is important to take into account a thorough history, clinical findings, biochemical testing including free T₄ and TSH, and imaging to make the diagnosis. The distinction between thyrotroph hyperplasia and thyrotroph adenoma is necessary because of the implications on treatment. Pituitary hyperplasia can be effectively treated with thyroid replacement, leading to regression of the mass on imaging. In cases of visual field deficits or ocular movement abnormalities due to nerve compression of the chiasm or cavernous sinus, surgical decompression might be necessary [5]. Although a rare entity, it is important to recognize primary hypothyroidism as a cause of thyrotroph hyperplasia in

order to spare patients unnecessary surgery. Compliance with thyroid hormone is also required in order to achieve normalization of TSH and regression of the pituitary mass. Serial imaging is an important means to monitor treatment response. Radiographic improvement of the sellar lesion should be noticeable within several months after normalization of TSH and serial imaging should be continued until resolution of the mass effect.

CONCLUSION

This case illustrates thyrotroph hyperplasia, reversible with thyroid hormone replacement, in a patient who initially presented with TSH > 2000. It is important to recognize primary hypothyroidism as a reversible cause of a pituitary mass.

Keywords: Hashimoto's thyroiditis, Hyperplasia, Hypothyroidism, Pituitary

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Author Contributions

Dennis Delasi Nyanyo – Conception of the work, Design of the work, Acquisition of data, Analysis of data, Interpretation of data, Drafting the work, Revising the work critically for important intellectual content, Final approval of the version to be published, Agree to be accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved

Amy Wheeler – Conception of the work, Design of the work, Acquisition of data, Analysis of data, Interpretation of data, Drafting the work, Revising the work critically for important intellectual content, Final approval of the version to be published, Agree to be accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved

Lisa B Nachtigall – Acquisition of data, Drafting the work, Final approval of the version to be published, Agree to be accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved

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Written informed consent was obtained from the patient for publication of this article.

Conflict of Interest

Authors declare no conflict of interest.

Data Availability

All relevant data are within the paper and its Supporting Information files.

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