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Pituitary Hyperplasia Due to Primary Hypothyroidism Mimicking Adenoma

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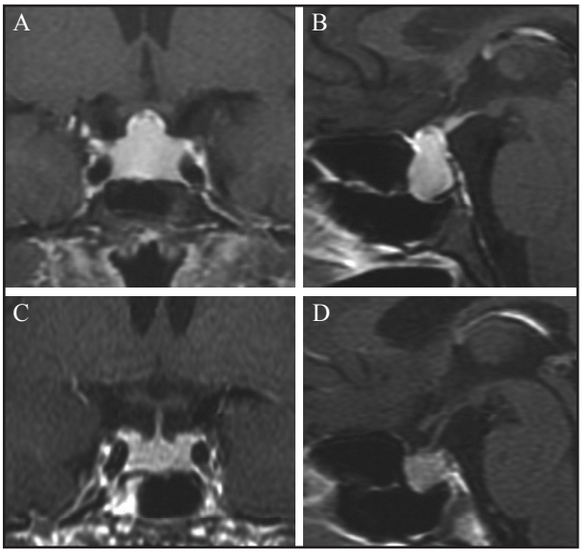

Figure 1: Initial coronal (A) and sagittal (B) gadolinium (Gd)-enhanced T1-weighted magnetic resonance imaging (MRI) scans show a homogenously enhancing intrasellar mass with suprasellar extension. After 5 months of thyroid hormone replacement, coronal (C) and sagittal (D) Gd-enhanced T1-weighted MRI scans reveal decrease in size of the pituitary mass.

Pituitary enlargement secondary to primary hyperthyroidism is rare, which results from the loss of thyroxine feedback inhibition and the subsequent overproduction of thyrotropin-releasing hormone (TRH), which thereby leads to hypersecretion of thyroid-stimulating hormone (TSH) and prolactin.[2,3] Radiographically, pituitary hyperplasia due to primary hypothyroidism can not be easily differentiated from an adenoma. Diminished T4 (thyroxine) with high TSH levels and occasionally high prolactin levels favor the diagnosis of pituitary hyperplasia due to primary hypothyroidism.

An 18-year-old female presented with menorrhagia, easy fatiguability, general malaise and swelling of the body. On examination, she had pallor and pretibial edema. Magentic resonance imaging (MRI) of brain revealed an intrasellar mass extending to suprasellar region, which was homogenously enhanced on gadolinium injection **(Figures 1A and 1B)**. Her hormonal profile showed extremely raised TSH (1011.9 μIU/ml, normal range 0.5-5.0 μIU/ml), low free T3 *i.e.* triiodothyronine (2.0 pg/ml, normal range 3.0-7.0 pg/ml), low free T4 (0.2 ng/dl, normal range 0.9-1.7 ng/dl) and high prolactin (76 ng/ml, normal range 4.9-29.3 ng/ml). Anti-thyroglobulin and anti-thyroid peroxidase antibodies were positive, indicating autoimmune thyroiditis. A follow-up MRI scan 5 months after institution of oral levothyroxine showed regression of the mass (Figures 1C and 1D). The hormone profile revealed normalised TSH (0.41μIU/ml), free T3 (5.7 pg/ml), free T4 (1.1 ng/dl) and prolactin (24 ng/ml).

It is known that loss of thyroxine feedback inhibition could accentuate a response leading to hyperplasia of thyrotrophs, simulating a pituitary adenoma. The incidence of associated hyperporlactinemia ranges from 1.1-40%, possibly due to lactotroph hyperplasia or mass

effects. The patients with pituitary hyperplasia due to hypothyroidism usually present with thyroid-related symptoms, rarely with neurological symptoms secondary to sellar expansion.[1] Despite recent progress in imaging techniques, this entity is difficult to differentiate from an adenoma. Re-evaluation after hormone replacement provides a definite diagnosis. Surgery should be reserved for the patients requiring decompression of optic chiasm or obtaining pathological diagnosis in the patients unresponsive to medical treatment. Thus, this case further advocates the need of thyroid function test in case of a pituitary mass to avoid unnecessary surgery.

References

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