International Journal of Clinical Science and Medical Research
ISSN(print): 2770-5803, ISSN(online): 2770-582X
Volume 05 Issue 01 January 2025
DOI: https://doi.org/10.55677/IJCSMR/V5I1-06/2025, Impact Factor: 7.606
Page No : 36-38

Pituitary Mass: Avoidable Mistakes and Essential Reflexes – Lessons Learned from a Clinical Case

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ABSTRACT

Pituitary masses require Standardized and medecine evidence-based management to prevent misdiagnosis and inappropriate management.This case describes a 41-year-old female presenting with depressive symptoms, hyperprolactinemia,a pituitary mass with visual impairment, initially misdiagnosed as a macroprolactinoma. The patient was initially referred for surgery; however, further investigations revealed severe primary hypothyroidism with secondary pituitary hyperplasia. Following thyroid hormone replacement therapy, the pituitary mass regressed, and symptoms resolved. This case highlights the importance of thorough evaluation and identifying reversible causes before proceeding with surgical interventions, thereby avoiding unnecessary procedures and associated risks.

Published Online : January 25, 2025

KEYWORDS:
Pituitary adenoma, Pituitary enlargement, Differential Diagnosis, Primary hypothyroidism , hyperprolactinemia ,Key reflexes,management

INTRODUCTION

The evaluation and management of a pituitary mass are critical aspects of neuro-endocrinology, requiring a precise and systematic approach to avoid diagnostic and therapeutic pitfalls. Prematurely attributing symptoms to a prolactinoma or overlooking systemic causes can lead to inappropriate treatments. This article highlights common mistakes and essential strategies for accurate diagnosis and effective management through the critical analysis of a clinical case.

CASE REPORT

We describe a 41-year-old non-pregnant, non-breastfeeding female patient with a 6-month history of fatigue and sluggishness, initially diagnosed as a depressive disorder and treated with antidepressants.

The patient consulted a general practitioner after developing amenorrhea and bilateral provoked galactorrhea. Laboratory tests revealed hyperprolactinemia at 69 ng/mL, with normal renal and liver function. Based on these findings, she was prescribed cabergoline at 0.5 mg/week.

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Cite this Article: Meryam Alahyane, Manar Bari, Sara ijdda, Sana Rafi, Ghizlane El Mghari, Nawal El Ansari (2025). Pituitary Mass: Avoidable Mistakes and Essential Reflexes-Lessons Learned from a Clinical Case. International Journal of Clinical Science and Medical Research, 5(1), 36-38

Two weeks later, the patient experienced acute headaches and blurry vision. Brain MRI revealed a 17 × 12.5 × 12 mm intrasellar mass with Isointense signals on T1 sequences, Hyperintense signals on T2 sequences,Moderate enhancement post-contrast injection.

The lesion extended superiorly, filling the optochiasmatic cisterns and abutting the optic chiasm. It was in close contact with the cavernous sinuses and intracavernous carotid arteries, with mild collapse of the right sellar floor. No ischemic or hemorrhagic lesion was noted (Figure 1).

Goldmann visual field testing showed: **Right eye**: Nasal step.**Left eye**: Enlargement of the blind spot.

The imaging was initially interpreted as a pituitary macroprolactinoma with visual impairment, and the patient was referred to the neurosurgery department for potential surgical management.

Further workup revealed:

- Adrenal insufficiency (8 a.m. cortisol: 4 µg/dL).
- Growth hormone (GH) deficiency (IGF-1: 48.6 ng/mL; normal: 75–249 ng/mL).
- Severe primary hypothyroidism (TSH: 195 mIU/mL, FT4: 0.4 ng/dL) with positive TPO antibodies and thyroiditis on cervical ultrasound.

Treatment included:

- Hydrocortisone (10 mg/day).
- Levothyroxine (2 µg/kg/day) with gradual dose escalation.

- Echocardiographic monitoring.
- Discontinuation of cabergoline and antidepressants.

After six months, the patient's thyroid function and prolactin levels normalized. Corticotropic and somatotropic deficiencies resolved, and follow-up MRI showed regression of the pituitary mass (Figure 2). Visual function also improved, and depressive symptoms were completely relieved.

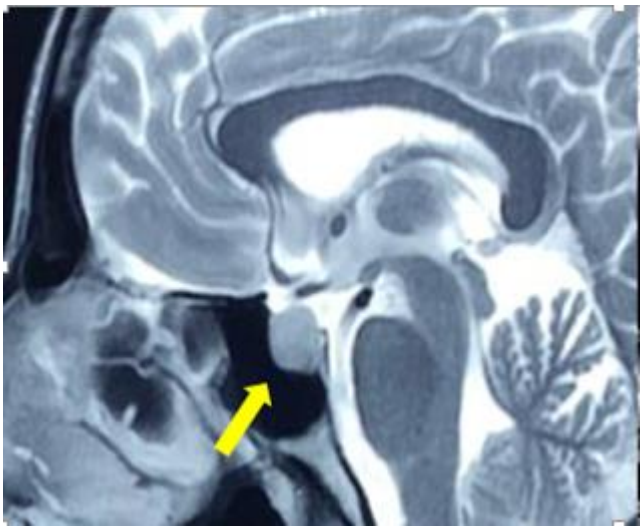

Figure 1: Sagittal T2-weighted MRI of the brain showing a 17 × 12.5 × 12 mm intrasellar mass, consistent with pituitary enlargement secondary to primary hypothyroidism, initially misdiagnosed as a pituitary macroadenoma.

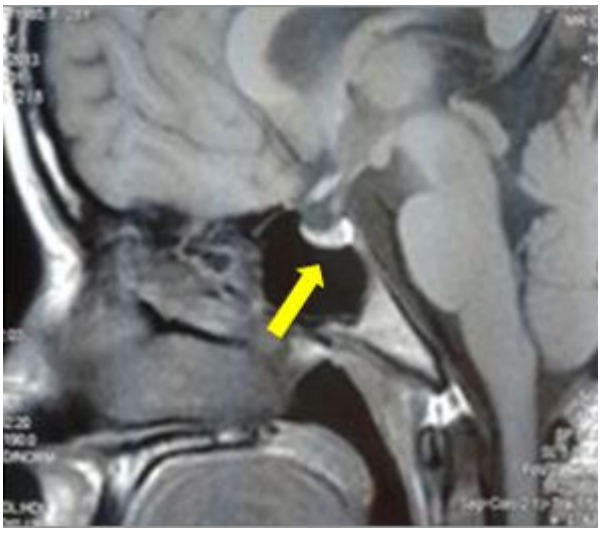

Figure 2: Sagittal T1-weighted MRI of the brain demonstrating regression of the pituitary mass after six months of thyroid hormone replacement therapy.

DISCUSSION

Through the mismanagament and the Good Practice Reflexes missed in this clinical case, we were able to draw the following lessons:

Lesson 1: Depression may signal an underlying endocrinopathy

Hypothyroidism is a well-established cause of depression. Potential mechanisms include:

- **Hypothalamic-Pituitary-Thyroid (HPT) Axis Dysregulation**: Thyroid hormone deficiency disrupts serotonin, dopamine, and norepinephrine balance, critical for mood regulation.
- **Reduced Neurogenesis and Synaptic Plasticity**: Hypothyroidism impairs hippocampal function, affecting mood and memory.
- **Neuroinflammation and Oxidative Stress**: Increased pro-inflammatory cytokines and oxidative stress contribute to depression.
- **Cerebral Hypoperfusion**: Reduced blood flow in brain regions involved in emotional processing.
- **HPA Axis Hyperactivity**: Elevated cortisol levels from HPA dysregulation exacerbate depressive symptoms.

Lesson 2: Pituitary mass may be secondary to untreated primary hypothyroidism

Chronic hypothyroidism leads to increased thyrotropin-releasing hormone (TRH) production, causing reactive pituitary hyperplasia. This condition mimics a pituitary adenoma but resolves with thyroid hormone replacement therapy.

Histopathological studies reveal that up to 91% of patients with longstanding hypothyroidism develop pituitary enlargement, while 12% progress to tumorous hyperplasia and 18.7% to adenoma [3]. Differentiating these conditions is crucial to avoid unnecessary surgery. Pituitary hyperplasia typically regresses with thyroid hormone therapy. Surgery should only be considered for masses unresponsive to treatment or with progressive complications, such as optic chiasm compression.

Lesson 3: Elevated prolactin with a pituitary mass is not always a prolactinoma

Hyperprolactinemia has diverse etiologies, including:

- **Physiological causes**: Pregnancy, lactation.
- **Systemic diseases**: Renal insufficiency, liver disease.
- **Primary hypothyroidism**: TRH-induced prolactin secretion.
- **Medications**: Dopamine antagonists, SSRIs, opioids.

In hypothyroidism, hyperprolactinemia typically resolves with thyroid hormone replacement. In cases of persistent elevation, consider a concomitant prolactinoma. PRL levels >200–250 ng/mL strongly suggest prolactinoma. Conversely, mildly elevated PRL (<100 ng/mL) with a large non-functioning pituitary adenoma often indicates stalk dysfunction.

The "hook effect" may lead to falsely low PRL levels in large adenomas and should be ruled out by reassessing PRL after serum dilution. [5,6]

CONCLUSION

Physiological pituitary hyperplasia should be included in the differential diagnosis of sellar masses, especially in untreated or undertreated hypothyroidism. Thyroid hormone therapy can reverse hyperplasia, avoiding unnecessary surgery and its complications, such as hypopituitarism or diabetes insipidus.

Take-Home Messages

- Screen for hypothyroidism in patients with depressive symptoms.
- Treat primary hypothyroidism with levothyroxine before considering surgery for a pituitary mass.
- Investigate hyperprolactinemia's etiology before initiating dopamine agonists.
- Rule out physiological, systemic, and pharmacological causes of hyperprolactinemia before diagnosing a prolactinoma
- Multidisciplinary discussion is crucial to avoid unnecessary invasive treatment and its complications in the management of pituitary masses.

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