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Biochemistry

Primary Hypothyroidism masquerading as Pituitary Adenoma

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ABSTRACT Pituitary masses are of great concern, and it is important to identify causes that could deter an invasive surgical intervention. We describe an unusual case of a 20-year-old male who was investigated for a new onset chronic intermittent headache. Brain imaging findings showed pituitary enlargement suggestive of pituitary macroadenoma. He was seen by a neurosurgeon for resection of that pituitary mass but surprisingly the thyroid function test was suggestive of primary hypothyroidism i.e. the markedly elevated thyrotrophin stimulating hormone (TSH) levels in the absence of clinical features of hyperthyroidism and low thyroid hormone levels .Patient did well with thyroid hormone replacement therapy and the repeat MRI following therapy showed significant resolution in 3 months of commencement.

KEYWORDS :

INTRODUCTION

Pituitary enlargement secondary to primary hypothyroidism is an uncommon occurrence and that the reactive pituitary gland enlargement is difficult to differentiate from inflammatory disorders or from a functional pituitary adenoma. [2]

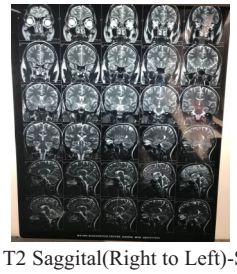
[2] Nicholas WC, Russell WFJ Miss State Med Assoc. 2000 Mar; 41(3):511-4

Case Report

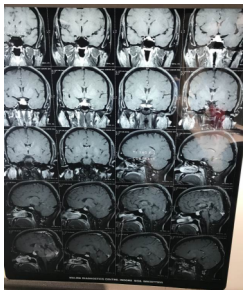
A 20-year-old male presented with intermittent headache of six to seven months duration, easy fatigability of about a year or so. Headache was described as bifrontal headache with no complaints of vomiting, photophobia or phonophobia that lasted few minutes to few hours and was episodic. He was given symptomatic treatment during his multiple clinical visits. His vitals were stable all throughout with pulse rate was 72 beats/minute and regular; blood pressure 120/80 mm hg. He was conscious, oriented, cranial nerves examination was normal and EOM full. There were no focal neurological deficits, deep tendon reflexes were normal. There were no signs of meningitis or of cerebellar involvement.

Soon, he was seen by Neurologist and a CT brain (plain) along with ophthalmologic examination was performed. CT Brain suggested a likelihood of pituitary macroadenoma. Imaging revealed expanded sella turcica with hyperdense pituitary lesion measuring about 1.1 x 1.1 cms suggestive likely of pituitary macroadenoma. There was slight suprasellar extension of about 1 to 2 mm. The optic chiasma appeared unremarkable. Inferiorly there was moderate bulge into the roof of sphenoid sinus. (Images not available)

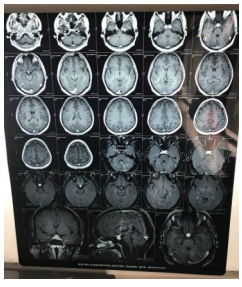
Patient was then advised an MRI and a reference was made to a Neurosurgeon. Patient was also subjected to endocrine workup of pituitary hormones. MRI embarked the lesion to be of size 16x16x14 mm and post contrast images revealed a small well defined solid mass lesion arising from the pituitary gland that shows small upper margin lobulation that extends in suprasellar cisterns and closely touching the inferior margin of optic chiasma. The lesion shows intense uniform contrast enhancement, the lesion appears as a primary pituitary adenoma with suprasellar extension as Figure 1.(a);1.(b) and 1.© respectively.



1.(a) T2 Coronal & T2 Saggital(Right to Left)-Showing well defined lesion in the sellar area with suprasellar extension marked with arrows(white).

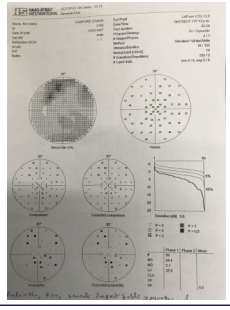


1.(b) T1 Coronal Post Contrast (fat suppressed) and T1 Saggital Post contrast (fat suppressed) Image.



1.(c)T1 axial Post Contrast images with selected coronal P.C and sagittal P.C showing the lesion vide supra.

Visual Acuity was 6/6 in both eyes. Slit lamp examination was normal. Fundus examination was normal. Perimetry was then advised which showed and as shown in figure 3.A.A repeat perimetry to look for visual field after 3 months was advised. VEP was done which was normal as shown in figure 3.B.



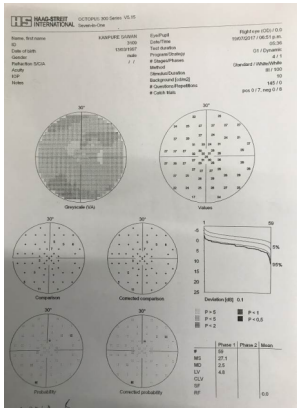


Figure 2.(a),2.(b) Perimetry.

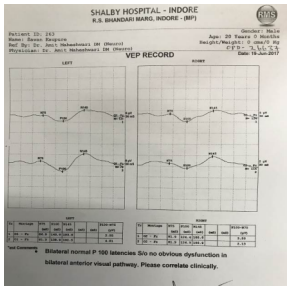


Figure 3. VEP.

Endocrine hormonal essay revealed S.Prolactin 13.3 ng/ml (range 3-20), GH 0.17ng/ml (range 0.05-3) and TSH of 416.22 mIU/ml (range0.4-4.2). Routine blood investigations were normal.

It was then that a diagnosis of primary hypothyroidism with pituitary hyperplasia was suspected. He was started on tablet L-thyroxine replacement doses. Dosage was slowly titrated to a maximum dose, starting with 75 mcg for first 5 days then 100mcg for next 5 days and then 150 mcg to be continued.

At three months follow-up, he was doing well and T3, T4 and TSH levels were in range of 2.32pg/ml, 0.58 ng/dl and 62.87mIU/ml respectively. Follow-up imaging was performed which showed significant resolution of the lesion as shown in figure 4.

Fig.4 MRI brain is normal. The Pituitary gland is slightly prominent in size and has a normal shape for age. Its vertical height is 10mm and is slightly enlarged for age and sex. No abnormal signal changes detected at present. As compared to the previous scan, there is significant reduction in size of the lesion seen at upper margins of pituitary gland.

Discussion

Reactive pituitary hyperplasia secondary to primary hypothyroidism is a benign cause of pituitary enlargement and has been described for more than a century. This enlargement is generally asymptomatic, however symptoms of pituitary mass such as compression of the optic chiasm, headache, amenorrhea and galactorrhea, due to hyperprolactinemia may be present. There is also report of intracranial hypertension.

Central hypothyroidism (CH) is a disease characterized by a defect of thyroid hormone production due to insufficient stimulation by TSH of an otherwise normal thyroid gland while in the setting of primary hypothyroidism, loss of thyroxine feedback causes hypothalamic production of thyrotropin-releasing hormone (TRH), which stimulates thyrotroph cells of the pituitary to produce TSH.[1]

[1] Joshi AS, Woolf PD. Pituitary hyperplasia secondary to primary hypothyroidism: a case report and review of the literature. Pituitary 2005;8: 99–103.

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In the above-mentioned case, there was regression of pituitary volume after 3 months of levothyroxine therapy confirmed the diagnostic hypothesis of pituitary hyperplasia resulting from primary hypothyroidism. The response time of thyroid hormone treatment required for regression of hyperplasia is still not well defined. There are reports of mass regression in 40 days (19), and there is still a report of dramatic pituitary regression with only 6 days of clinical treatment (13).

Transfotoidal surgery reports for treatment of pituitary mass subsequently confirmed as a result of primary hypothyroidism (14) reinforce the importance of the evaluation of thyroid hormones and TSH in the investigation of pituitary volume increase, preventing an inadvertent clinical or surgical treatment.