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Unusual Presentation of Primary Hypothyroidism

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Authors' contributions

This work was carried out in collaboration between all authors. All authors read and approved the final manuscript.

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Case Study

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ABSTRACT

Primary hypothyroidism may present with myriad of unusual presentation apart from typically described signs and symptoms. We report an unusual case of primary hypothyroidism clinically presenting as acute psychosis and radiologically mimicking as pituitary macroadenoma. A 19-year-old female presented with h/o abnormal behaviour in the form of auditory hallucination, fearfulness, loss of memory and inability to recognise family members six months back. She also gave h/o multiple joint pain, easy fatigability, facial puffiness, somnolence, progressive weight gain, constipation and cold intolerance for similar duration. MRI brain revealed enlarged pituitary, while the thyroid function analysis pointed towards primary hypothyroidism. Patient improved with LT4.

Keywords: Hypothyroidism; psychosis; pituitary hyperplasia.

1. INTRODUCTION

Acute psychosis is an uncommon presenting feature of overt hypothyroidism occurring in 5% to 15% of myxedematous patients [1]. Enlarged pituitary gland due to primary hypothyroidism is also a rare finding & this reactive enlargement of the gland may not be easily differentiated from functional pituitary adenomas [2,3]. We present a case of primary hypothyroidism presenting as acute psychosis with imaging suggestive of sellar mass with suprasellar extension.

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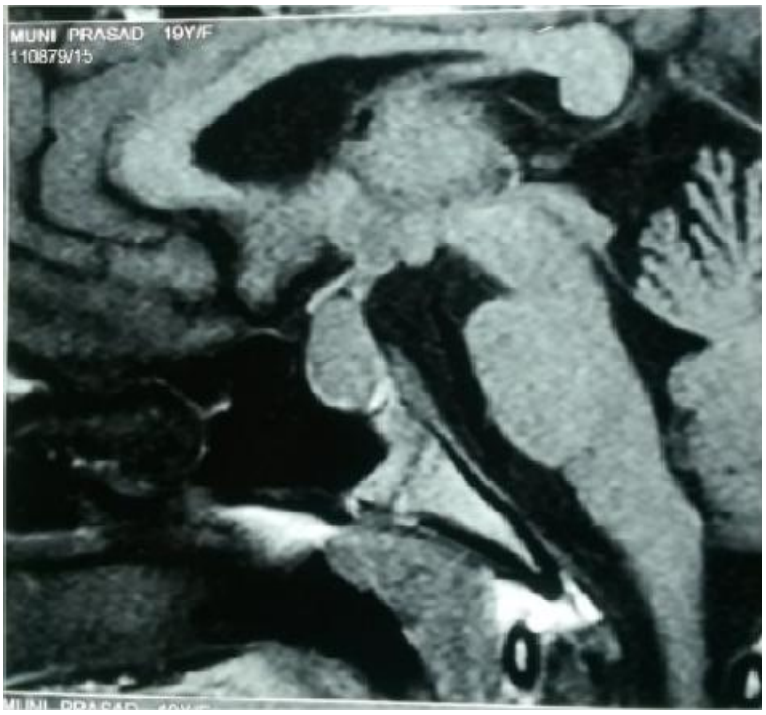
2. CASE

A 19-year-old female presented to a psychiatrist with abnormal behaviour in the form of auditory hallucination, fearfulness, loss of memory and inability to recognise family members six months back. She was evaluated in a local hospital and a CT scan was done which suggested a sellar mass. Further, MRI pituitary (Fig. 1) was done which showed sellar mass of 0.9×1×1.46 cm^3 with suprasellar extension with compressed bright spot without involvement of adjacent structures. She was put on Tab. Risperidone but no significant improvement of symptoms occurred, and subsequently referred to our endocrine clinic for evaluation. On detailed examination, she also gave h/o multiple joint pain, easy fatigability, somnolence, facial puffiness, constipation, and cold intolerance of 9 months duration. There was no history of headache, visual disturbance, seizure, polyuria, polydipsia, loss of consciousness or galactorrhoea. Her menstrual cycles were regular and appetite was normal.

On examination, She was awake, alert, and conversant but with notable auditory hallucinations during the examination. Pulse rate was 56 beats/minute and regular and blood pressure was 124/96 mm of Hg without postural drop. Thyroid gland was normally palpable. Her neurological examination was essentially normal except for the delay in relaxation phase of her deep tendon reflexes. Other systems were normal. Routine biochemical and haematological investigations were normal, except low hemoglobin (11.4 gm / dL). Her baseline anterior pituitary function tests were normal except for raised thyroid stimulating hormone (TSH) (>100 mIU/L, range 0.40-4.0) with a low total T4 (0.31 µg/dL, range 6-12.0). On further evaluation, we found anti-TPO antibody to be positive (267IU/dl). Based on these findings the most probable possibility of autoimmune thyroiditis and primary hypothyroidism with probable pituitary hyperplasia was made. However a diagnosis of pituitary macroadenoma was also kept in mind. Her antipsychotics were stopped after admission to the hospital. She was started on tablet levothyroxine 50 mcg per day (low dose to avoid aggravation of psychosis) and was gradually uptitrated to a dose of 88 mcg daily. Her abnormal behaviour subsided in a matter of 7-10 days and other hypothyroid symptoms improved over 1-2 months. At six months of follow up, she was doing well. Her T4 and TSH levels were in normal range. Follow-up MRI (Fig. 2) pituitary suggested regression of the mass (0.86×0.97×0.83 cm^3).



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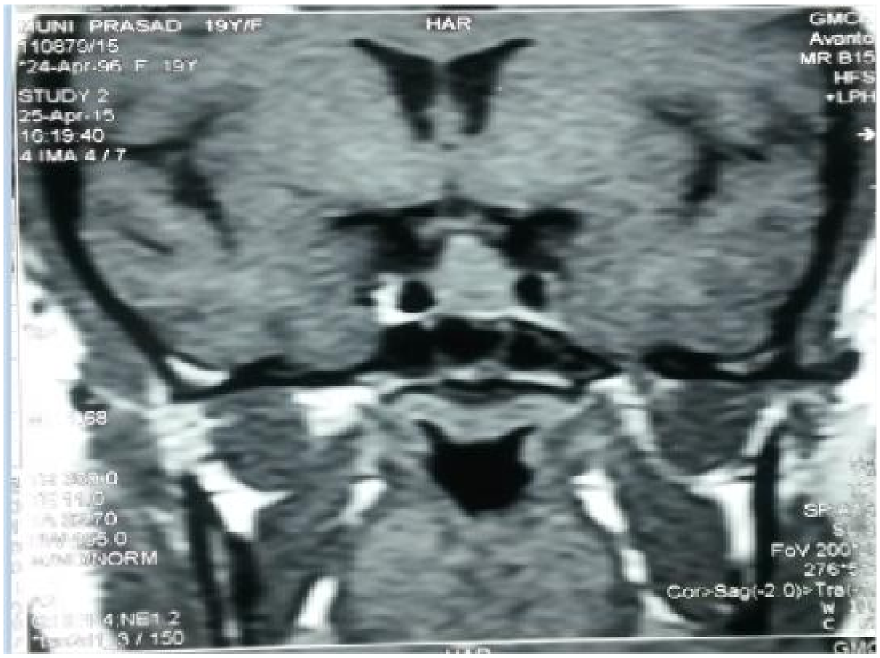
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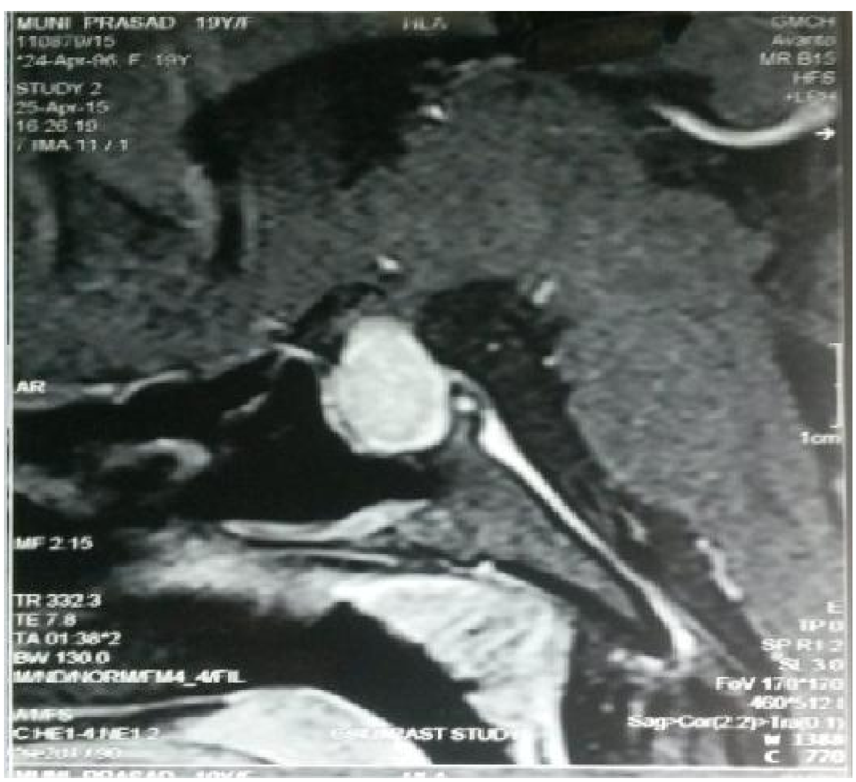
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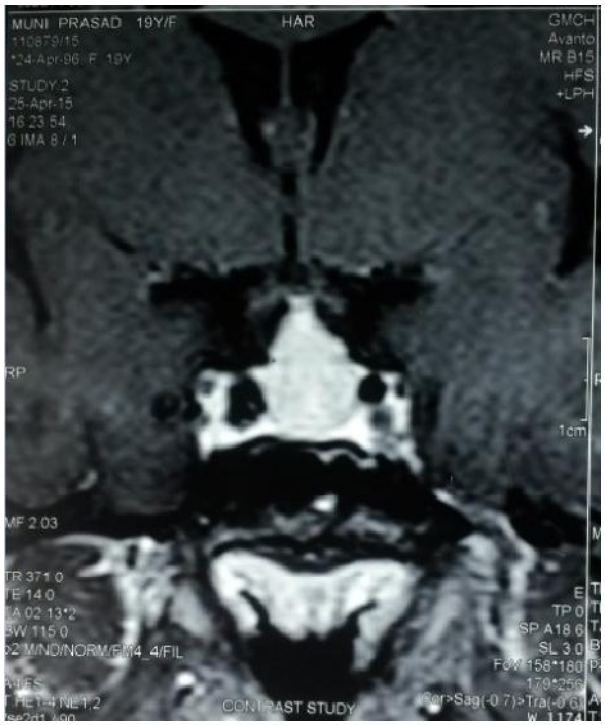
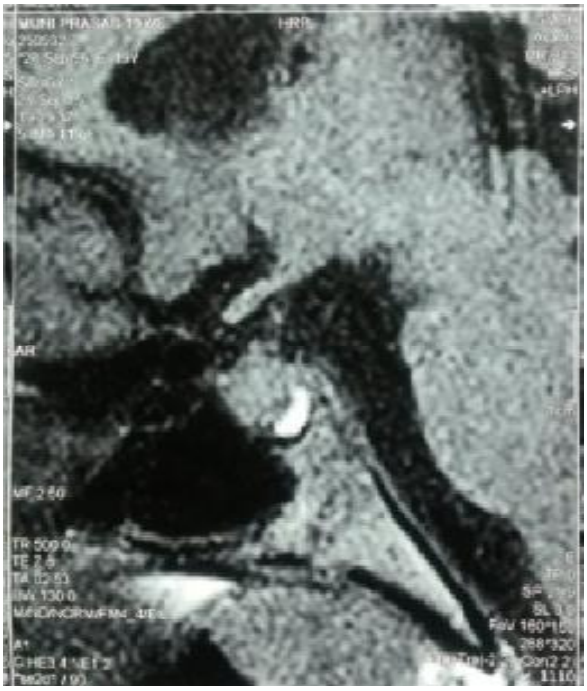


(d)

Fig. 1. MRI pituitary at presentation T1W1 pre (a, b) and post contrast (c, d) images in sagittal and coronal sections



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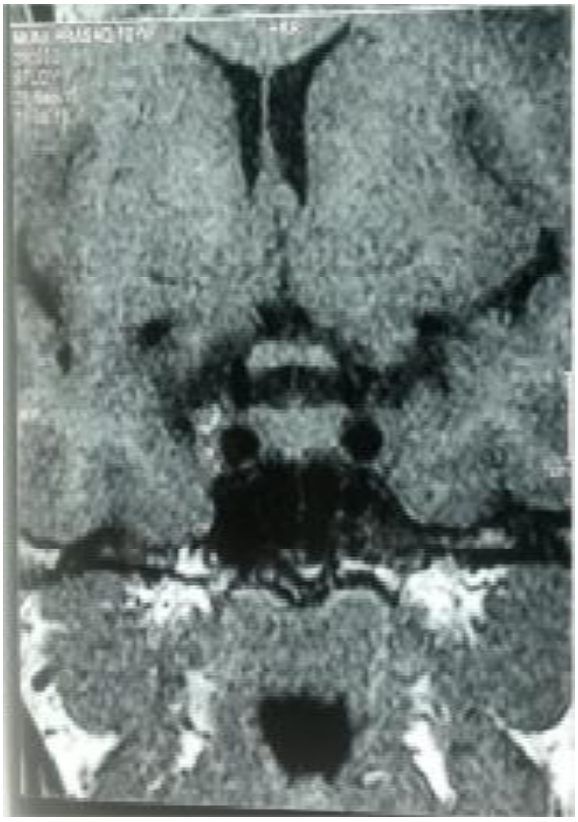

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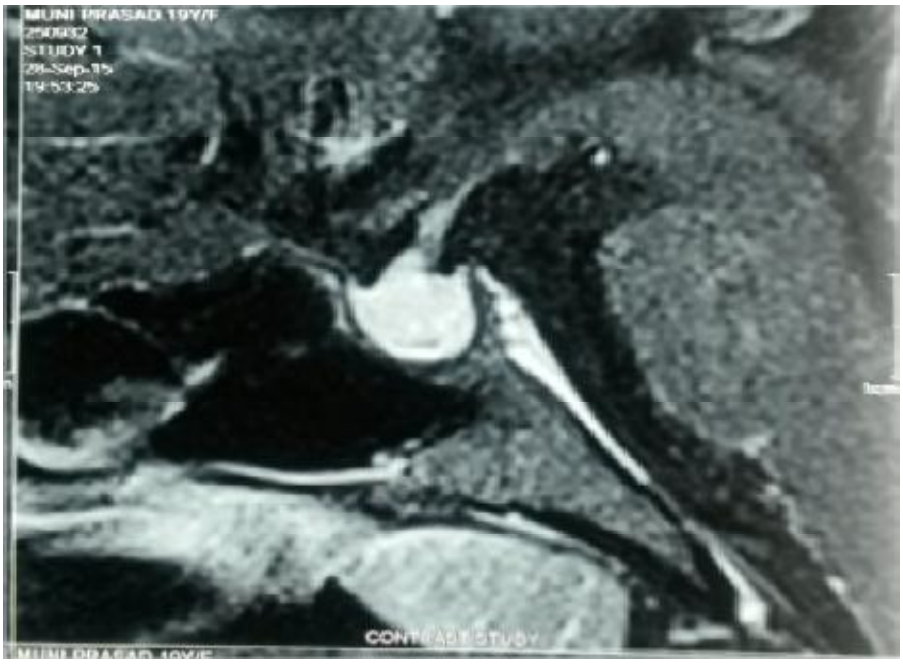

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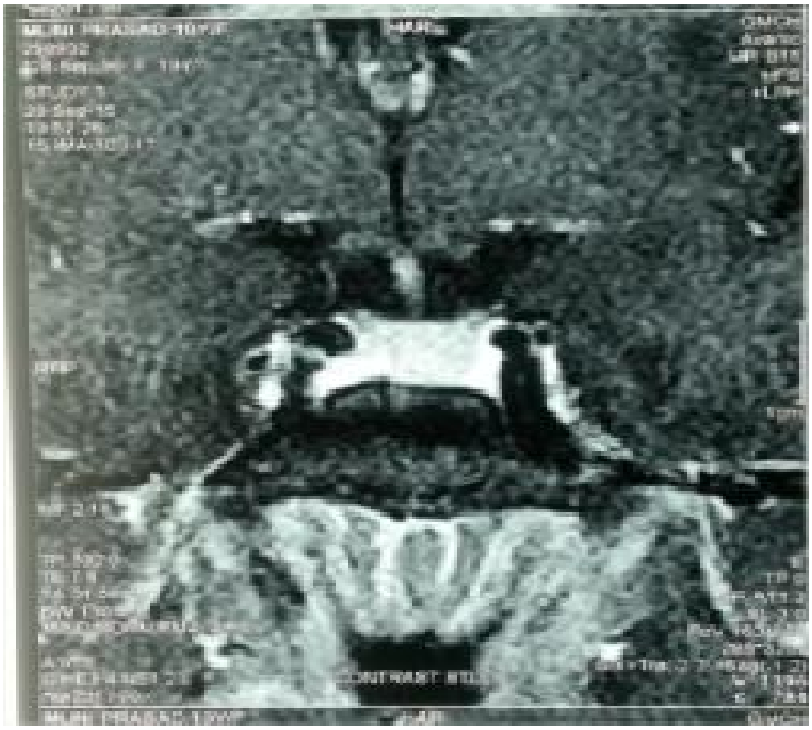

(d)

Fig. 2. MRI 6 months after LT4 replacement T1W1 pre (a, b) and post contrast (c, d) images in sagittal and coronal sections

3. DISCUSSION

Primary hypothyroidism presenting as acute psychosis along with a mass in pituitary gland is a rare entity with occasional case reports. Identification of this entity is important so that unwanted removal of the enlarged gland by the neurosurgeon can be avoided.

Our patient came to us with abnormal behaviour along with hypothyroid features; findings that favoured the diagnosis of acute psychosis with primary hypothyroidism, [4] a diagnosis further supported by positive hormonal analysis and imaging study. Earlier reports have suggested that cases with markedly elevated TSH and low thyroid hormone levels without clinical features of hyperthyroidism, one should strongly consider pituitary enlargement secondary to primary hypothyroidism [5]. Such cases of pituitary hyperplasia are due to enlargement of thyrotroph cells owing to absent negative feedback. The hormonal profile of such patient can be easily reversed with thyroid hormone therapy.

Both, pituitary thyrotroph and lactotroph cells are stimulated [6] by increased Thyrotropin releasing hormone (TRH) levels which in turn is due to loss of negative feedback from thyroxine, the level of which is low [7]. This leads to increase TSH secretion and prolactin. Literatures report the occurance of pituitary hyperplasia in patients with primary hypothyroidism at a frequency of 25-81% [8]. Yamada et al. [9] demonstrated a correlation between the serum TSH level and increase in size of sella turcica. Recently, Khawaja et al. [10] found that 70% patients with TSH levels ≥ 50 mIU/L had pituitary enlargement.

Such patients present with psychosis after months to years following onset of physical symptoms [11]. Both subclinical and clinical hypothyroidism are known to be associated with thyroid disorders [12]. Brain utilizes thyroid hormone differently than other organs. Thyroid hormone receptors are located in large numbers in amygdala and hippocampus and influence the intracranial neural networks [13]. These receptors are known to be highly sensitive to thyroid hormones and in hypothyroid cases, the low levels of hormones are shuttled to cater to the brains need, [14] thereby explaining the later onset of psychological symptoms.

Identification of this entity is important as a simple approach such as thyroxine replacement can lead to complete cure as evidenced by regression of pituitary enlargement on MRI in over 85% cases [10]. This change has been noted as fast as within 1 week to upto several months [15,16,17,18,19,20]. Regression of hypertrophied tissues depends on the mechanism of inflammatory circle by interleukins. So this time is changeable depending upon the depressor agents and the receptors of the hypertrophied tissue. We could not perform a repeat MRI before 6months in our case by which time complete regression had occurred.

4. CONCLUSION

This paper reports a rare case of a patient with symptoms of acute psychosis, fatigue and enlargement of pituitary gland, suggesting the occurance of primary hypothyroidism. It also emphasizes the fact that levothyroxine replacement leads to complete regression as evident by follow up MRI. This simple watchful approach by the treating physician can save the patient from undergoing unwanted pituitary surgery.

CONSENT

All authors declare that written informed consent was obtained from the patient (or other approved parties) for publication of this case report and accompanying images.

ETHICAL APPROVAL

It is not applicable.

COMPETING INTERESTS

Authors have declared that no competing interests exist.

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