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BMH Med. J. 2018;5(1):20-23 **Case Report**

An Interesting Case of Hyopnatremia

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Abstract

Symptomatic hyponatremia is a common case of altered sensorium in elderly patients admitted to medical ward or intensive care units. Our patient was taking thyroid replacement since 18 months. He had clinical features of hypogonadism with recurrent hyponatremia necessitating multiple hospital admissions. Due to multiple hormonal deficiencies viz., Thyroid, Gonadotropin and ACTH, he was evaluated for pituitary disorders and was diagnosed to have a pituitary macro adenoma on MRI brain which was later excised through transphenoidal endoscopic approach resulting in full recovery and treated with hormone replacement.

Keywords: hyponatremia, pituitary macroadenoma

Introduction

Hypopituitarism is a clinical syndrome of deficiency in pituitary hormone production [1,2]. This may result from disorders involving the pituitary gland, hypothalamus, or surrounding structures. Panhypopituitarism [3] refers to involvement of all pituitary hormones however, only 1 or more pituitary hormones are often involved, resulting in isolated or partial hypopituitarism. If pituitary failure is acute, the patient will be acutely ill and presents with hypotension and shock, or even coma, however if the onset is chronic and the pituitary deficiency is mild the patient may express only fatigue and malaise along with many changes [4] in the skin, hair and nails as a result of the absence of pituitary hormone action on these sites.

Case report

Middle aged male presented to us with a history of fever, vomiting and loose stools of 10 days duration following which he developed altered sensorium of 7 days duration. He has been on treatment for hypothyroidism since 18 months with 50mcg thyroxin tablet daily. He had pallor of skin since 18 months. Following vomiting and loose stools three months ago he was treated for hyponatremia at local hospital. He was not suffering from diabetes mellitus, coronary artery disease, pulmonary tuberculosis, seizure disorder or any drug allergies. There was no family history of hypothyroidism and parents were non-consanguineous.

He was conscious, disoriented, dehydrated, moderately built and nourished. Pallor of skin and mucous membrane were present, axillary and pubic hair were sparse. He was drowsy with GCS-13

(E3V4M6), Pupils were normal reacting to light equally on both sides, he was moving all four limbs and had delayed relaxation of ankle jerks bilaterally, no signs of meningeal irritation, optic fundi were normal. His visual field charting was normal.Other systems were with in normal limits. He had normal body temperature, Blood pressure was 150/80mm Hg in supine position in right upper limb, pulse rate 60/min regular with normal volume. His respiratory rate was 18/min. His visual field charting was normal. Routine blood investigations showed Hb - 11.1 gm/dl, Total WBC – 6,000 cells/cmm. ESR: 34mm/1st hour. Chest X-ray was with in normal limits. RFT: Normal, LFT: Normal. Urine routine: Normal. Serum sodium: 108meq/L, Urine osmolality: 278 mosm (Normal range: 500-800), Serum osmolality: 241.3mosm (Normal range: 290-320), Urine sodium excretion: 100meq/L (Normal range: 40-220).

He was treated with hypertonic (3%) saline infusion at the rate of 20ml/hr for 3 days (daily correction not more than 10-12meq/l). Three days later, he became better but hyponatremia persisted with serum sodium -120meq/l. We further evaluated the patient to rule out the endocrinopathies especially a pituitary disease. Cortisol (8am): 3.85 microgram/dl (Normal range: 8.7-22.4). 4pm: 3.24microgram/dl (Normal range: 8.7-22.4), ACTH level: 9.47 pg/dl (Normal range: 9-52). Thyroid profile during this admission was not done because he was on thyroxin.

With past history of hypothyroidism, and clinical features of hypogonadism, and secondary hypoadrenalism with low levels of ACTH and cortisol, we further proceeded to evaluate with MRI brain to rule out pituitary abnormality.

MRI brain showed intrasellar mass with suprasellar extension and mass effect on optic chiasma and hypothalami, with no obvious cavernous sinus invasion bilaterally, suggestive of pituitary macroadenoma (**Figure 1**).

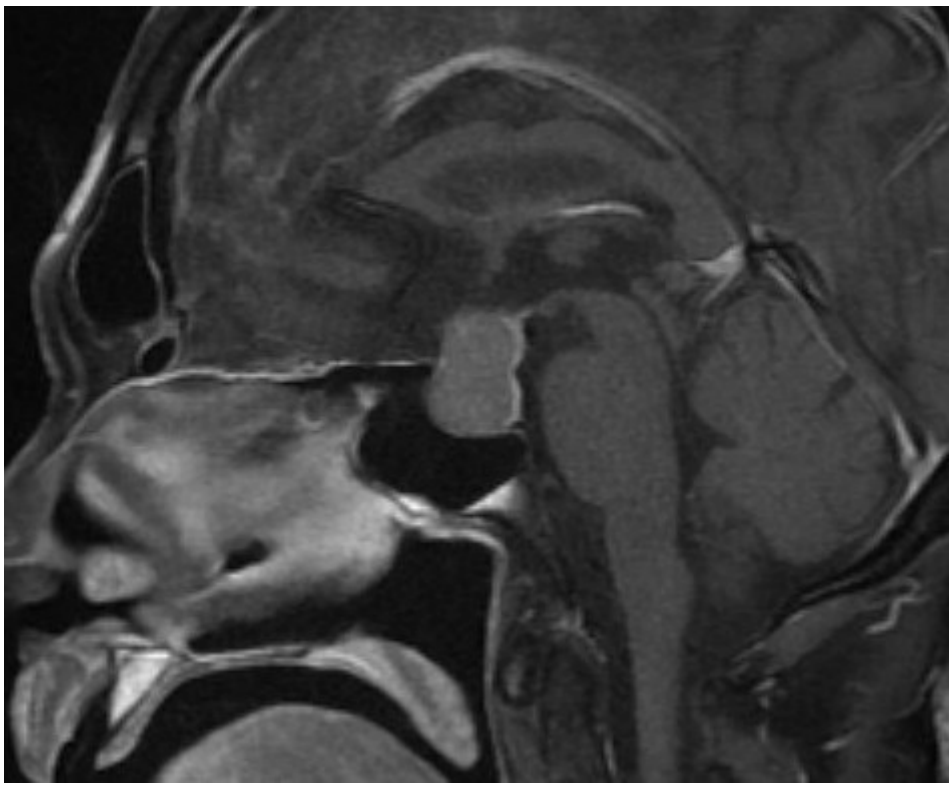

Figure 1: MRI showing intrasellar mass with suprasellar extension and mass effect on optic chiasma and hypothalami, no obvious cavernous sinus invasion bilaterally, suggestive of pituitary macroadenoma

Patient was referred to ophthalmology and neurosurgery department for further evaluation. Ophthalmology evaluation showed normal optic fundi and visual fields bilaterally. After evaluation by ENT surgeon he was given a course of oral antibiotics for maxillary sinusitis. After controlling the sinusitis he was taken up for transnasal-transphenoidal endoscopic assisted excision of pituitary macro adenoma by the neurosurgeon. Postoperative period was uneventful.

Patient discharged on thyroxine 50 mcg OD and prednisolone and on re-view after 1 month, he was found to be improving and was asymptomatic. His serum electrolytes were normal and thyroid hormone levels were normal.

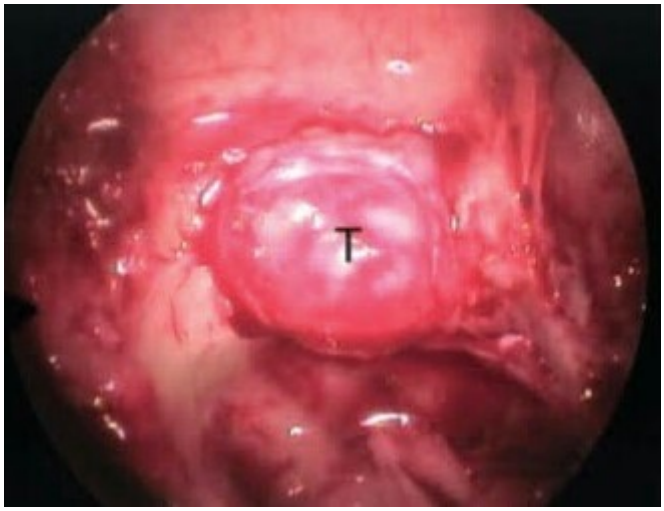

Figure 2: Endoscopic view of sellar content after removing duramater (T: Tumour)

Discussion

The sellar region is a site of various types of tumors. Pituitary adenomas are the most common. They arise from epithelial pituitary cells and account for 10-15% of all intracranial tumors. Tumors exceeding 10 mm are defined as macroadenomas, and those smaller than 10 mm are termed microadenomas.

Pituitary macroadenomas are benign epithelial neoplasms composed of adenohypophysial cells. Primary malignant tumors of the pituitary are extremely rare. The monoclonal nature of most pituitary tumors suggests that they arise from a mutated pituitary cell. However, the exact pathophysiologic/molecular mechanisms leading to the development of pituitary adenomas remain unknown. Some pituitary tumors may occur as part of a clinical syndrome. In multiple endocrine neoplasia type 1 (MEN 1), an autosomal dominant genetic disorder, pituitary adenomas (most often prolactinomas) occur in association with tumors of the parathyroid and pancreatic islet cells.

Patients with malfunctioning pituitary tumors are often asymptomatic and are discovered incidentally during cranial imaging procedures performed for other reasons. This is true for both microadenomas and macroadenomas. Tumors that cause symptoms are usually large space occupying macroadenomas which compress nearby neurologic structures. Clinical features include headache, visual field defects, visual loss and extra ocular nerve palsies. Pituitary insufficiency also may result from destruction of normal pituitary tissue.

In our case he was receiving treatment for hypothyroidism with thyroid hormone replacement before presenting with acute diarrheal illness. He had profound hyponatremia, possibly due to central adrenal insufficiency and central hypothyroidism, apart from loss of water and electrolytes due to diarrhea. Sparse pubic and axillary hair was suggestive of central hypogonadism. He had no visual field defects indicating normally functioning optic chiasma and optic tract. MRI brain confirmed the presence of intrasellar macroadenoma which was resected through transsphenoidal approach. The presentation of pituitary macroadenoma was chronic in nature with vague symptoms with profound hyponatremia and features of hypogonadism.

References

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