



CASE OF HYPONATREMIA IN NON-FUNCTIONING PITUITARY MACROADENOMA

General Medicine

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ABSTRACT

Patients with recurrent episodes of hyponatraemia should be evaluated with fasting serum Cortisol and imaging of the brain if necessitated when other systemic causes of hyponatraemia are ruled out. Non secretory pituitary adenomas can compress the normal pituitary gland causing hypopituitarism. Here is a patient 45-year-old male from Kannur, Kerala who presented with recurrent episodes of transient loss of consciousness, generalized weakness with symptoms of dehydration and persistent hiccups for 2 years. Patient was detected to have low sodium levels in different hospitals for 2 years and supportive therapy was given after which patient had felt symptomatically better for few days. The patient now presented to the hospital again with complains of generalized weakness and persistent hiccups. Patient was evaluated with random cortisol which was low and was admitted and evaluated further. The 8AM cortisol levels were low and was evaluated further with Syntropic stimulation test to differentiate central vs peripheral cause of hypocortisolism. MRI Brain with contrast was suggestive of Pituitary Macroadenoma 16x14x14 mm in Sella extending to suprasellar cistern. (Histopathology being suggestive of Non-secretory Macroadenoma).

KEYWORDS

Hyponatremia, Pituitary adenoma, Cortisol, ACTH (Adrenocorticotropic hormone), Syntropic stimulation test

INTRODUCTION

The prevalence of nonfunctioning pituitary adenomas varies between 60 and 100 cases per million population. It has a bimodal peak incidence between the ages of 25–45 years and 60–70 years and the incidence rate are 1.02–1.08 per 100000. There is an equal incidence among both genders.

Hyponatraemia is defined as a serum sodium level less than 135 mmol/L. Severe hyponatremia is when serum sodium is below 125 mmol/L, which is associated with increased morbidity and mortality.

These lesions are usually found incidentally (mainly microadenoma) or diagnosed based on symptoms and signs of anterior pituitary hormone deficiency and compressive symptoms such as headache and visual field defects.

Nonfunctioning pituitary macroadenomas generally present with visual disturbances, headache, and symptoms due to anterior pituitary hormone deficiencies.

This case report is about an atypical presentation of a nonfunctioning pituitary macroadenoma in which the patient presented with recurrent episodes of loss of consciousness and recurrent persistent hiccups.

Case Study

45-year-old male patient from Kannur, Kerala presented with recurrent episodes of brief loss of consciousness, generalized weakness with symptoms of dehydration and persistent hiccups for 2 years and history of decreased libido for 6 months. There was no history of vomiting, loose stools, or history suggestive of seizures, fever, headache, or decreased urine output. Patient also had decreased appetite, recent significant weight loss. Patient was detected to have low Sodium levels in different hospitals which patient had visited over the time and supportive therapy was given.

There was no history of intake of any regular medication including steroid medications. Clinically patient had stable vitals and systemic examination revealed bitemporal hemianopia on visual field examination using confrontation method. Other systemic examination was normal.

Patient blood investigations revealed hyponatremia, as the reports are mentioned below. And random cortisol levels were done to look for hypocortisolism, which was low (35.2 nmol/L). Patient was evaluated further with syntropic stimulation test which showed increase in levels of cortisol after the test which was suggestive of ACTH deficiency.

MRI Brain with contrast revealed Pituitary Macroadenoma 16x14x14 mm in Sella extending to suprasellar cistern (Probably Non-secretory Macroadenoma)

Table 1: Laboratory Investigations:

TEST	24/3/2022	6/4/2022	Post Syntropic Stimulation Test
S. Sodium		131 mmol/L	
S. Potassium		4.6mmol/L	
S. Cortisol	Random-35.2nmol/L (Low)		343.3nmol/L
8 AM Cortisol		102.1nmol/L (Low)	
Free T4		0.551ng/dL(Low)	
S. Testosterone		1.25ng/dL(Low)	
S. Prolactin		30.7ng/mL(Elevated)	

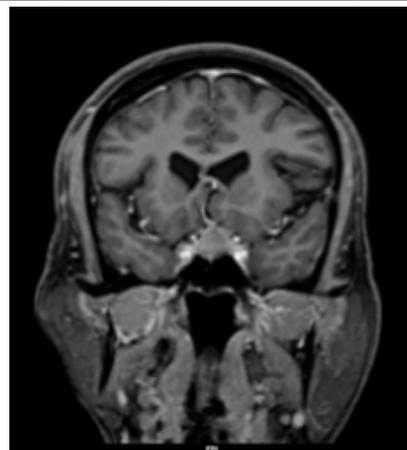


Figure 1: Coronal Section of Brain with Pituitary Protocol

MRI report- Altered signal intensity of well-defined lesion is noted in the sella extending to suprasellar cistern measuring 16 x 14 x 14 mm (TR, CC, AP). It appears isointense to grey matter on T1 and T2W sequences and shows heterogeneous enhancement on post contrast T1W sequences. Superiorly the lesion is extending upto the optic chiasm. Laterally it is abutting the cavernous segment of internal carotid artery bilaterally. Posterior pituitary is displaced superiorly. Pituitary stalk is displaced laterally towards right side. A heterogeneously enhancing lesion in the sella, extending to suprasellar cistern. Features suggestive of Pituitary macroadenoma seen.

Management

Patient was initiated on IV Normal Saline, Tab Prednisolone 5MG OD, Tab Thyronorm 75 MCG OD, Oral Calcium supplements. Patient underwent Excision of lesion (trans-sphenoidal) in MIMS, Kerala and is currently asymptomatic.

Biopsy of lesion was suggestive of Pituitary adenoma positive for FSH, synaptophysin.

CONCLUSION

Although nonfunctioning pituitary macroadenomas generally present with symptoms and signs of anterior pituitary hormone deficiencies and mass effect on adjacent structures, they can also have unusual presentations. Cyclical vomiting and severe hyponatremia are atypical presentations of nonfunctioning pituitary macroadenomas.

DISCUSSION

Nonfunctioning pituitary adenomas commonly appear as pituitary macroadenomas. Their symptoms and signs are usually due to anterior pituitary hormone deficiency and mass effect on adjacent structure, especially on optic chiasma, occurring in 60%–80% of cases. There can be mild hyperprolactinemia (<100 mg/ml) due to the compression of pituitary stalk by the tumour interrupting the descending dopaminergic effects. In this case, the patient presented with atypical symptoms of nonfunctioning pituitary macroadenoma, though he had biochemical evidence supportive of a pituitary macroadenoma. He presented with recurrent episodes of loss of consciousness. He complained of generalized body weakness and reduced appetite, which might be nonspecific symptoms associated with hypoadrenalism.

Hyponatremia due to hypopituitarism is usually due to secondary hypoadrenalism rather than central hypothyroidism. Hyponatremia due to secondary hypoadrenalism is caused by impaired electrolyte-free water excretion in the absence of normal cortisol activity in the kidney. Also, there is increased secretion of arginine vasopressin, a secondary ACTH secretagogue, which results in urinary concentration and can further impair hyponatremia. Unlike in primary hypoadrenalism, in secondary hypoadrenalism, since there is no mineralocorticoid deficiency, it is unlikely that patients develop hyperkalemia. Our patient had severe hyponatremia with minimal hyponatremic symptoms due to its chronic nature.

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