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Case Reports

A Case of Pituitary Macroadenoma with Recurrent Hyponatremia-Hiding Behind Diuretic Use

Dr. Anupama Sahu, Dr. Mohammad Zeya Ansari*, Dr. Nibedita Mishra, Dr. Kunal Priyadarshi, Dr. Murtaza Ahmed Khan, Dr. Sarita Kumari, Dr. Aastha Verma

Department of General Medicine, Tata Main Hospital, Jamshedpur, PIN code-831001, Jharkhand, India

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ABSTRACT

The incidence of pituitary macroadenoma associated with hypopituitarism is relatively rare and uncommon. As the importance of research in macroadenoma of the pituitary gland increases, we see a concomitant rise in reporting of such cases. In this clinical case, we report a 76-year-old man, with a frequent history of hospital admissions due to various symptoms resulting from hyponatremia, the most alarming of which was an episode of transient loss of consciousness. He was on oral hypoglycemic drugs, an angiotensin receptor blocker, a beta blocker and a diuretic which led to a suspicion of diuretic induced hyponatremia in previous two instances. It was his third hospitalization with transient unconscious state with severe hyponatremia but without a diuretic which made us suspect a different etiology and so he was investigated further. A strong clinical suspicion, blood investigations and neuroimaging confirmed it to be a case of pituitary macroadenoma with secondary adrenal insufficiency. He was managed conservatively and discharged with the appropriate treatment.

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INTRODUCTION

Clinicians confront cases of hyponatremia in day-to-day practice. Common causes of hyponatremia include diuretics, gastrointestinal fluid loss, renal failure, heart failure, decompensated liver cirrhosis, syndrome of inappropriate antidiuretic hormone secretion (SIADH). Adrenal insufficiency secondary to pituitary gland adenomas leading to hyponatremia is an uncommon presentation. Most of these tumours are benign and grow at a slow rate. Classification is typically based on tumor size or the specific pituitary cell type of origin. According to size, pituitary adenomas are categorized as microadenomas (<10 mm), macroadenomas (≥10 mm), and giant adenomas (>40 mm). [1,2]

Functionally, pituitary adenomas are divided into functioning and nonfunctioning types. Functioning adenomas secrete excessive amounts of single or multiple hormones of the anterior pituitary, leading to distinct clinical syndromes depending on the hormone involved.

In contrast, nonfunctioning adenomas are non-secretory but may give rise to symptoms secondary to mass effect, such as compression of the normal pituitary gland, resulting in hypopituitarism, or adjacent structure involvement (e.g., optic chiasm) leading to visual disturbances. [1]

Management of pituitary adenomas requires a multidisciplinary approach, typically involving endocrinologists, ophthalmologists, and neurosurgeons. Despite extensive research, the pathogenesis of pituitary adenomas remains incompletely understood. Most cases are sporadic, though a minority are associated with familial syndromes. In an Icelandic cohort study of 410 pituitary adenoma cases, 43% were nonfunctioning, 40% secreted prolactin, 11% secreted growth hormone (GH), and 6% secreted adrenocorticotropic hormone (ACTH). [3]

Pituitary adenomas rarely involve genetic mutations, yet familial cases constitute about 5% of all tumors. Several gene mutations have been linked to tumorigenesis, including those involved in cell cycle regulation, signal transduction, and transcriptional control.[4]

***Corresponding author:**

Dr. Mohammad Zeya Ansari,
Department of General Medicine,
Tata Main Hospital, Jamshedpur,
PIN code-831001, Jharkhand, India
Email: dransari24@gmail.com

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Clinical Case:

A 76-year-old male was admitted with complaints of sudden loss of consciousness lasting for around 10 minutes at home. There was no history of tonic-clonic movements, up-rolling of eyeballs, tongue bite, or involuntary urination or defecation. The patient had pre-existing diabetes mellitus and hypertension and was on regular treatment. His medications included metformin 1 gm twice, gliclazide 80 mg twice, voglibose 0.3mg twice, telmisartan 80 mg once, metoprolol 50 mg twice, and chlorthalidone 12.5 mg once per day. He had been admitted twice previously for giddiness with hyponatremia secondary to diuretic with acute gastritis and vomiting, which had improved with 3% sodium chloride infusion and oral salt supplementation and proton pump inhibitor. As he was on a diuretic, investigations revealed hypo-osmolar (270 mOsm/kg) hypovolemic hyponatremia. After improvement he was discharged with advice of avoiding diuretic. In this hospital admission, he was conscious and oriented with Glasgow coma scale equal to 13 but reported generalized weakness making him stand with support only. General clinical and systemic examinations did not reveal abnormal findings. His vital parameters were as follows: pulse rate 82/ min, blood pressure 166/82 mmHg, random blood sugar 130 mg/dL, and pulse oximetry showed presence of 98% oxygen on room air. Hematological parameters were within normal limits except for mild anemia (Hb 10.3 g/dL). He had good control of diabetes with random blood sugar below 120 mg/ dl and Hba1c 5.6%. Serum creatinine was 1.17 mg/dL and vitamin B12 level was 1053 pg/mL. Laboratory investigations showed serum sodium 125 mEq/L, potassium 4.1 mEq/L, urinary sodium 68 mEq/L, and urinary potassium 24 mEq/L. He had was low serum osmolality (264 mOsm/kg) with no features of fluid overload, so it was euvolemic hypo-osmolar hyponatremia. As there was no history of gastrointestinal fluid loss and there was no drug being used that could cause dyselectrolytemia, a possibility of syndrome of inappropriate antidiuretic hormone (SIADH) was suspected. In that sequence, thyroid function test and serum cortisol levels were sent. He had low morning cortisol (0.39 µg/dL) and thyroid stimulating hormone (TSH= 0.27 µIU/mL) with T3 and T4 being 6.01 ng/mL and 1.04 µg/dL respectively. Further endocrine evaluations were done in the line of hypopituitarism which demonstrated serum prolactin–3.03 ng/mL, luteinizing hormone (LH) -1.69 IU/L, follicular stimulating hormone (FSH) 2.03 IU/L and testosterone 9 ng/dL. (table 1 below)

Table 1: Endocrinological blood reports

HORMONES	MEASURED LEVEL	NORMAL
Prolactin (PRL)	3.03 ng/ml	2-18 ng/ml
Cortisol (M)	0.39 mcg/dl	5 to 25 mcg/dL
LH	1.69 IU/L	1.8 to 8.6 IU/L
FSH	2.03 IU/L	1.5 to 12.4 IU/L
Testosterone	9 ng/dl	264 to 916 ng/dL
TSH	0.27 µIU/mL	0.27 – 4.2 µIU /mL

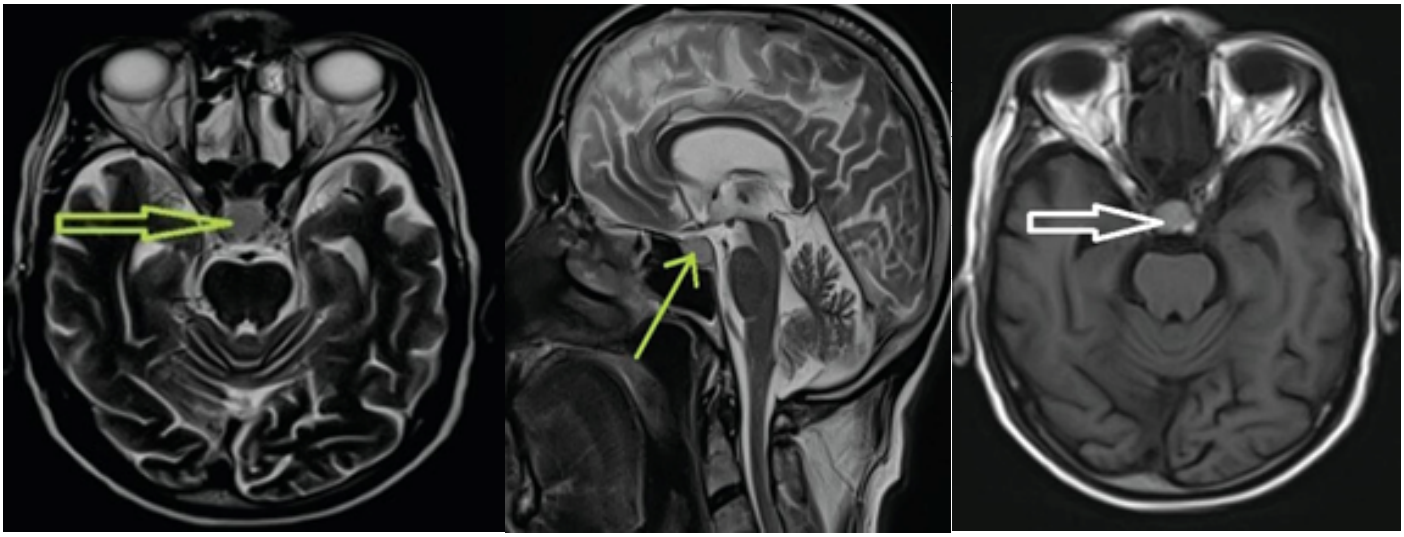


Fig 1,2,3: MRI brain images showing pituitary macroadenoma measuring approximately measuring 0.91 x 1.28 x 1.31 cm

Cardiac evaluation revealed normal electrocardiogram while echocardiography showed grade I left ventricular diastolic dysfunction only. A Holter monitoring was done for 24 hours to rule out arrhythmias which was reported normal. He also had non-contrast computed tomogram of head through emergency which showed age-related cerebral atrophy with chronic ischemic changes. An electroencephalogram (EEG) was done which revealed normal brain activity. Neck vessel doppler revealed no significant luminal narrowing. In relation to the low TSH and serum cortisol levels, a pituitary pathology was suspected, and hence contrast-enhanced magnetic resonance imaging (CEMRI) of the brain was done which showed age-related cerebral atrophy with chronic microvascular ischemic changes and a pituitary macroadenoma measuring 0.91 x 1.28 x 1.31 cm (fig 1,2,3). There were no visual field defects on perimetry. He was started on intravenous hydrocortisone 100 mg stat and 50 mg 8 hourly. We could see his serum sodium normalizing with it. After 3 days, he was administered 50 micrograms of oral thyroxine. A neurosurgical opinion was taken and was advised for conservative replacement therapy. The final diagnosis was pituitary macroadenoma with hypopituitarism with secondary adrenal insufficiency with diabetes mellitus and hypertension. The patient was discharged in stable hemodynamic condition on amlodipine, prednisolone, and levothyroxine. On follow-up in the medical outpatient department, the patient remains clinically stable, asymptomatic, and without recurrence of hyponatremia.

Discussion:

Pituitary macroadenomas are benign neoplasms arising from the anterior pituitary gland and may present with varied clinical manifestations depending on their size, hormonal activity, and the degree of compression of surrounding structures. Although visual disturbances, headache, and features of hormonal hypersecretion or deficiency are the typical presenting symptoms, atypical manifestations such as recurrent hyponatremia or transient loss of consciousness may also occur, leading to diagnostic challenges. [2, 5]

In the present case, the patient exhibited recurrent episodes of hyponatremia without any overt neurological deficits or visual symptoms. He was initially managed as diuretic-induced hyponatremia being on chlorthalidone for essential hypertension. As a result, there was a delay in proper etiological diagnosis. It manifests often as SIADH and only a

strong clinical suspicion after stopping the diuretic for nearly two weeks can give us correct diagnosis of true SIADH. The underlying mechanism for hyponatremia in pituitary macroadenoma is multifactorial and may include secondary adrenal insufficiency, hypothyroidism, or the syndrome of inappropriate antidiuretic hormone secretion (SIADH) [6,7]. In this patient, markedly low cortisol and thyroid hormone levels were suggestive of hypopituitarism leading to secondary adrenal insufficiency and hypothyroidism, which may cause impaired free water clearance and subsequent dilutional hyponatremia. [6]

The pituitary macroadenoma identified on MRI likely resulted in compression of normal pituitary tissue, leading to hormonal deficiencies rather than hypersecretion, as evidenced by the low levels of ACTH-dependent cortisol and gonadotropins. The absence of elevated prolactin levels ruled out a functioning prolactinoma. Conservative management, consisting of glucocorticoid, testosterone (tab testosterone undecanoate 40 mg twice daily), and thyroid hormone replacement, effectively corrected the biochemical abnormalities and prevented further recurrence of hyponatremia.

This case highlights the importance of considering pituitary pathology as an underlying cause of recurrent or unexplained hyponatremia, particularly in elderly patients. Early recognition of secondary endocrine insufficiencies is crucial, as timely initiation of appropriate hormone replacement can result in rapid clinical and biochemical improvement [8]. A multidisciplinary approach, involving internal medicine, endocrinology, and neurosurgery, is suggested for optimal management and long-term follow-up of such patients.

Conclusion:

We need to be vigilant while evaluating a case of recurrent hyponatremia as a manifest cause of hyponatremia might hide the actual and unexpected etiological diagnosis. Besides, cases with diuretic-induced hyponatremia need to be under supervision and regular follow-ups to avoid unwanted poor outcomes and complications Moreover, in patients presenting with recurrent hyponatremia or recurrent vomiting, neuroimaging and comprehensive hormonal evaluation should be undertaken to exclude pituitary pathology and guide timely management.

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