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PITUITARY MACROADENOMA PRESENTING AS RECURRENT HYPONATREMIA : A RARE CASE PRESENTATION

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ABSTRACT

Hyponatremia, defined as plasma Na^+ < 135 mM, is a commonly occurring electrolyte imbalance seen in almost 22 % of hospitalized patients. Need prompt diagnosis and management as it is potentially life threatening condition. Presentation of pituitary adenoma varies but microadenoma will be generally asymptomatic and macroadenoma will present with compressive symptoms. We report a 66 year old male patient who presented with recurrent hyponatremia secondary to non-functioning pituitary macroadenoma rather than compressive feature, which recovered after surgical removal of adenoma.

INTRODUCTION:

Hyponatremia occurs due to disruption of water balance. Hypernatremia always denotes hypertonicity but hyponatremia can be associated with low, normal, or high tonicity. Dilutional hyponatremia, by far the most common form of the disorder, is caused by water retention.[1-3] Successful treatment of hyponatraemia depends upon accurate diagnosis of the underlying etiology of hyponatraemia; again, published data shows that in many cases, clinicians do not order the appropriate tests to enable them to arrive at the correct diagnosis.[4]

Having recurrent hyponatraemia is troublesome, as it can lead to neurological damage and potentially fatal too if timely and definitive diagnosis is not made [5]. Different central nervous system lesion like head injury, pituitary adenoma can cause hyponatremia .[6]

But pituitary macroadenoma presenting as recurrent hyponatremia before other feature like headache, visual problem is quite rare. Here, we report a case of an elderly male patient presenting with recurrent hyponatremia due to pituitary macroadenoma.Case History:

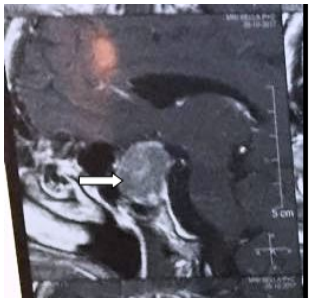
A 66 year old male presented with altered senosium since last 2 days. There was no history of any past major illness and any drugs intake or operative procedure. On admission patient s BP was 150/90 mmHg, pulse was 75 bpm, afebrile. He didn't have any, loss of axillary or pubic hair, cushingoid appereance, hyperpigmentation or features suggestive of generalized myxedema. CVS, R/S and abdominal examination was unremarkable. CNS examination - GCS score 11/15, no signs of meningeal irritation, fundus and CSF examination were normal. On enquiring about previous medical history patient had three similar episodes of recurrent hyponatremia during last one year for which he was hospitalized and managed with high salt diet & 3 % NaCl.

He did not have any sign of dehydration or fluid overload hence diagnosis of euvolemic hyponatremia was made and the patient was further investigated to find out the cause for the recurrent hyponatremia.

Biochemical studies in a patient with hyponatremia.

Plasma	D1	D2	D4	D8	Noramal rang
Hb (g/dl)	10				12-16
Tlc (/cu mm)	8500				4000-11000
Glucose (mg/dl)	82				70-150
Creatinine	0.9				0.4-1.5
Bun (mg/dl)	17.4				15-40
Na (mEq/L)	117	112	134	123	136-145
k(mEq/L)	3.6	3.5	4.4	3.6	3.5-5.6
Cl (mEq/l)	78	80	100	84	97-107
Osmolality (mosm/kg.H20)	252				275-295
TSH (ug/dl)	2.93				0.45-4.5
Total T4(ulU/ml)	4.39				4.87-11.72
Cortsisol (Ug/dl)	0.42				5-23
FSH (ulU/ml)	2.46				1.4-15.4
LH (ulU/ml)	1.20				1.2-7.8
Prolactin (ng/ml)	7.74				3.46-19.4
GH (ng/ml)	<0.05				0.05-3

Because of recurrent hyponatremia, we did pituitary hormonal profile which showed panhypopitutarism (low cortisol and TFT), so we did MRI brain, which revealed sellar and suprasellar mass lesion of 3.5 × 3 ×2.4 cm involving pituitary stalk with mass effect over optic chiasma with minimal heterogenous contrast enhancement .



MRI BRAIN – Figure no.1

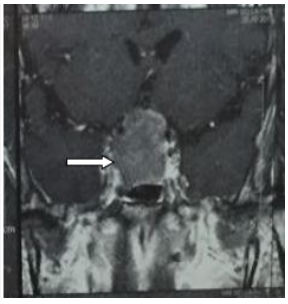


Figure no .2

He was thus diagnosed as a case of panhypopituitarism due to pituitary macroadenoma with hyponatremia and treated with T. thyroxine 75 µg OD along with T. prednisolone 5mg OD. The patient underwent surgical debulking after neurosurgery consultation. Subsequently patient was followed up and showed remarkable improvement in serum Na level and discharged with Na 136 mEq/l and did not develop hyponatremia thereafter. At monthly follow-up, the patient is doing well on tablet thyroxine and tablet predisolone.

DISCUSSION:

Hyponatremia has wide range of causes. It is of utmost importance to identify the underlying cause of hyponatremia and give a benefitting treatment which will prevent its recurrence as severe hyponatremia whose consequences varies from altered sensorium to coma and death if not treated correctly. In the majority of subjects with hyponatremia has defect in renal water excretion which is usually due to an inability to suppress ADH secretion [7] ACTH deficiency may present clinically with hyponatremia , but the

underlying mechanisms of hyponatremia in ACTH deficiency differ somewhat from those of primary adrenal insufficiency.In secondary adrenal insufficiency, hyponatremia results from impaired excretion of water loads, whereas in primary adrenal insufficiency, renal salt wasting is commonly seen, adrenal mineralocorticoid release is affected[8] and hyperkalemia may be seen.[9] Clinically, patients with partial ACTH deficiency are euvolemic, which can cause clinicians to confuse ACTH deficiency with SIADH.

As Cortisol is an inhibitor of ADH secretion, low level of cortisol leads to increased ADH level and causes dilutional hyponatremia [10]. Thus, the benefit of corticosteroid replacement is thought to be mainly due to suppression of ADH. Hyponatremia is also seen in hypothyroidism due to inability to excrete free water, and also they do not dilute urine maximally after a water load which could be due to increased ADH levels and Decreased GFR [11]. Due to limited resources measurement of ADH level in our patient was not done. To make diagnosis of SIADH exclude hypoadrenalism and hypothyroidism. This may be the case with our patient who was treated earlier for the episode of hyponatremia and correct diagnosis was not made. This highlights the importance of following the specific protocol while making a diagnosis of hyponatremia. It is often seen that patients with subclinical hypopituitarism develop hyponatremia in the presence of a precipitating factor [12]. Our case apparently had no symptoms related to hypopituitarism. Our patient was treated earlier with salt replacement and normal saline without making correct diagnosis patient recovered well from those episodes. This suggests that apart from dilutional hyponatremia a component of dehydration or sodium deficiency due to salt deficiency may be an additional mechanism responsible for the hyponatremia in this case. The differentiation between the following conditions: the SIADH, acute ACTH deficiency, fluid overload and the cerebral salt-wasting syndrome is of utmost importance because management of each differs [13]. Our case shows the various issues with regard to the workup and management of hyponatremia. The first episode is the time when adequate laboratory studies should be obtained so that a diagnosis can be reached expeditiously. A detailed and conclusive research regarding endocrine control mechanisms causing hyponatremia in the setting of hypopituitarism is lacking and may be a subject of future studies

CONCLUSION:

Pitutary hormonal profiling is must in all patients presenting with recurrent hyponatremia.

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