Severe Hyponatremia as an Uncommon Presenting Feature of Pituitary Macroadenoma

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Abstract

Hyponatremia is a common electrolyte disturbance, but less commonly utilized and require a thorough evaluation to unmask etiology. It has variety of causes and is rarely due to hypopituitarism. Hyponatremia is a very early complication of pituitary tumor. Here, we report a case, who presented to us with hyponatremia and eventually thorough work-up led us to a diagnosis of Non-functioning pituitary macroadenoma.

Introduction

Hyponatremia is serum sodium level of <135 mEq/L. It can lead to symptoms like decreased ability to think, irritability, lethargy, headache, nausea, and poor balance.[1] Severe symptoms include confusion, seizures, and coma.[2,3] The cause of hyponatremia is classified according to person's fluid status into hypo-,eu- and hypervolemic hyponatremia. Hypovolemic hyponatremia can result from diarrhoea, vomiting, diuretics and sweating. Euvolemic hyponatremia is seen in polydypsia, hypothyroidism, adrenal insufficiency and SIADH. Hypervolemic hyponatremia is seen in congestive heart failure, liver cirrhosis and renal failure. Pituitary macroadenomas' (>10 mm) are the most common cause of hypopituitarism, and in the majority of cases they are non-secretory adenomas.[4] A pituitary adenoma usually presents with bitemporal hemianopia due to compression of optic chiasma. Other symptoms are headache, psychiatric symptoms like depression, anxiety, or symptoms due to hyper-pituitarism if secretory adenoma is present. Secondary adrenal insufficiency can occur due to a pituitary tumor and thus result in hyponatremia.

Case Report

Our case was a 60 year old right handed male, resident of Nagore (Rajasthan, India), retired postmaster, who presented with chief complaints of multiple episodes of vomiting since 15 months, episodes of giddiness, blackouts, fatigue and lethargy since 4 months, and single episode of loss of consciousness 1 month back. Vomiting was non-bilious, non-projectile, 1 to 2 episodes per day, associated with nausea and relieved by medications. Giddiness was associated with episodes of blackout. There was an episode of loss of consciousness which lasted a few minutes, without any premonitory symptoms, involuntary movements or confusion after the episode. No history of headache, neck pain, altered sense of smell, visual symptoms, facial numbness or weakness, decreased hearing, loss of taste or altered taste, difficulty in swallowing, breathing. No history of trauma, unknown bite, vaccination, muscular weakness, fever, diarrhea or severe vomiting. He was non-alcoholic, non-smoker, vegetarian with normal bowel and bladder habit without any history of diabetes, hypertension, or any significant drug history.

On general physical examination,

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On general physical examination, patient was drowsy but easily arousable, oriented to time, place and person. He was afebrile (98.2 °F), hypotensive (90/60 mmHg), had tachycardia (120 beats per minute), with normal respiratory rate (16 breaths per minute). Conjunctivae and tongue were dry with reduced skin turgor. Axillary and pubic hair were sparse. There was no palor, icterus, cyanosis, clubbing, lymphadenopathy or pedal edema. On nervous system examination, higher mental functions were normal, visual field reduced on confrontation test, plantar response was extensor bilaterally, with normal power at all joints and normal deep tendon reflexes. Examination of sensory system, cerebellar signs, cranium spine and other cranial nerve were normal. Differential diagnosis kept were metabolic encephalopathy, vertebro-basilar insufficiency, chronic meningitis and vestibular disorders.

In routine investigations, complete blood count, liver and renal functions were normal (Hemoglobin-11.3 g/dL; Total leucocyte count- 8,540 / cumm; Platelet- 1.42 lacs/mL; Total bilirubin- 1.1 mg/dL; SGOT/PT- 34/18 U/L; ALP-113 U/L; LDH- 432 U/L; Urea- 16mg/dL; Creatinine- 0.93 mg/dL). However, uric acid was raised (7.9 mg/dL). Amongst electrolytes, there was hyponatremia (112 mEq/L) with normal potassium (4.6 mEq/L) and chloride (88 mEq/L). Calcium (7.9 mg/dL) and phosphate (4.6 mg/dL) were normal. Random blood sugar was 62mg/dL. Arterial blood gas analysis was normal (pH- 7.45, pCO2- 26.8, pO2- 104, HCO3- 22.5, SO2- 98.4%). Serum and urine osmolality were also normal (288.5 and 373 mosm/L respectively). To find cause for loss of sodium, 24 hr urine electrolytes were estimated which revealed increase loss of sodium in urine (224 mEq/L). Uptill now, provisional diagnosis of hypovolemic hyponatremia due to urinary loss of sodium resulting in metabolic encephalopathy was kept.

Patient was started with normal saline infusion and oral salt supplementation. Evaluating the case further, serum aldosterone levels were sent, which came out to be low (1.74 ng/dL). Evaluating other adrenal hormones, serum cortisol and testosterone levels also came out to be low (4.3 µg/dL and 5.75 ng/dL respectively), thus indicating hypoadrenalism. To differentiate between primary and secondary hypoadrenalism, ACTH stimulation test was done, which resulted in increase in serum cortisol levels (upto 18.11 µg/dL), thus indicating adrenal failure due to suppressed pituitary-adrenal axis with normal adrenal reserve. Other pituitary hormones were almost normal (TSH- 0.82 µU/mL; LH- 1.26 mIU/mL; FSH- 5.06 mIU/mL; Prolactin- 5.03 ng/mL) and FT3- 1.63 ng/dL, FT4-0.47 ng/dL indicated mild hypothyroidism. These investigations indicated a non secretory pituitary tumor. So, MRI Brain with Sellar Cuts with contrast was obtained, which revealed 28 x 24 mm sellar and suprasellar mass which was not differentiable from pituitary and caused upward deviation of diaphragmatic sella with compression of optic chiasma. Visual Acuity in-RIGHT EYE was, Distant-6/36, after correction- 6/18; and in LEFT EYE- Distant- 6/9, after correction-6/6. Perimetry revealed bitemporal constriction of visual fields. Thus a final diagnosis of Non functioning pituitary macroadenoma with secondary adrenal insufficiency with hyponatremia resulting in metabolic encephalopathy was kept. Patient was discharged in stable condition on Tab. Levothyroxin, Tab.Dexamethasone and Tab. Fludrocortisone. Patient was advised for surgery by neurosurgery department and is under regular follow up.

Discussion

Hyponatremia is often present in patients with intracranial pathology or those recovering from neurosurgery.

Syndrome of inappropriate anti-diuretic hormone secretion (SIADH) and cerebral salt wasting syndrome (CSWS) are the main causes.[5-8] Apart from these two, pituitary mass leading to secondary hypoadrenalism and hypothyoroidism can also lead to hyponatremia. Distinguishing these conditions is important as management is different for each of them. Hyponatremia in SIADH is caused by excessive ADH resulting in free water reabsorption. Mechanism of natriuresis in this condition is attributed to an increase in the glomerular filtration rate or a reduction in the tubular reabsorption of sodium. Diagnosis of SIADH is made in the presence of a plasma osmolality <275 mOsm/Kg with urine osmolality >100 mOsm/kg, clinical euvolaemia and an elevated urinary sodium excretion in the presence of a normal salt and water intake. Hypothyroidism, hypoadrenalism and diuretic use must be excluded prior to making a diagnosis of SIADH.

Cerebral Salt Wasting Syndrome (CSWS) results from excessive natriuresis in patients with intracranial disease. The mechanism underlying this natriuresis is unknown but has been proposed to involve the release of natriuretic factors (atrial natriuretic peptide, brain natriuretic peptide, c-type natriuretic peptide and oubain-like peptide) together with decreased sympathetic input to the kidney.[6,8,9] Cortisol is an inhibitor of ADH secretion, thus secondary hypoadrenalism due to pituitary non secretory macroadenoma, results in increased ADH secretion and consequently there is decreased excretion of water by the kidney and hyponatremia.[10,11]

Our patient was a classical example of secondary hypoadrenalism leading to hyponatremia which is suspected less often.

Conclusion

Hyponatremia is a very important diagnostic tool, which, if evaluated completely, can unmask serious underlying etiology, allowing us to manage properly and without delay.

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