An Uncommon Case of Abnormal Behaviour

Karthick Ganesh\textsuperscript{a}, Baiju Faizal\textsuperscript{a}, M G K Pillai\textsuperscript{a}

\textsuperscript{a} Department of General Medicine, AIMS, Kochi\textsuperscript{*}

\textbf{CASE REPORT}

41 year old male with no known comorbidities had an episode of vomiting and irrelevant talk 3 weeks back, for which he was taken to a local hospital for evaluation. He had an episode of GTCS on the way and was found to have hyponatremia (Na 110 meq), which was corrected and he was discharged as symptoms improved. He had two more admissions for altered behaviour, each time had hyponatremia which was corrected and he was discharged as symptoms subsided. 3 days back, behavioural problems had resumed in the form of irrelevant speech. He had become completely mute for a day.

When he presented to us, he was completely mute, conscious with spontaneous limb movements. He was afebrile and hemodynamic parameters were normal. Blood sugars were also normal. CNS examination was done as far as possible- all DTR were elicited, bilateral plantars were flexor, there was no neck stiffness and fundus was normal. Cardiovascular, respiratory and abdominal examinations were all normal.

ECG and Chest X Ray was normal. At this point we considered an encephalitis and a metabolic encephalopathy. We first evaluated for metabolic causes. Complete blood count, inflammatory markers, liver and renal functions were normal. Metabolic parameters showed hyponatremia (Na 125.6 mEq/L) with normal potassium, Magnesium, calcium, phosphorous and ammonia. Keeping in mind the history of recurrent hyponatremia we further evaluated the hyponatremia to reveal urine sodium of 65mmol/L, serum osmolarity 261mOsm/kg, urine osmolarity of 487.4mOsm/kg, serum uric acid 4.4mg/ dl, TSH 0.2uIU/ml, free T4<0.4ng/ml and T3<1pg/ml. Thus a central hypothyroidism was unearthed. We went ahead with an MRI brain and demonstrated a small anterior pituitary. We estimated anterior pituitary hormones to show a low testosterone, low prolactin and a low serum leutinizing hormone with normal FSH and ACTH. We ruled out underlying seizures (non convulsive status) with an EEG and diagnosed Hypopituitarism. We started the patient on 100mcg of eltroxin after confirming hemodynamic stability with an ECHO and saw dramatic improvement in symptoms in two days of starting therapy. He regained speech and was fully functional. He was discharged and is doing well on follow up, hyponatremia and other symptoms have not recurred.

\textbf{ABSTRACT}

We report the case of a 41 year old male with repeated admissions of symptomatic hyponatremia (seizures, behavioural abnormalities) who presented to us in a mute state. Evaluation of the hyponatremia revealed hypopituitarism and his symptoms completely subsided on therapy with eltroxin.

\textbf{Keywords:} Abnormal behaviour, Seizures, Hypopituitarism

\textsuperscript{*}See End Note for complete author details

Corresponding Author:
Dr. M G K Pillai, Professor, Department of General Medicine, AIMS, Kochi.
E-mail: kar1411.g@gmail.com
DISCUSSION

Hypopituitarism is a clinical syndrome of deficiency in pituitary hormone production. It can be Inherited/acquired and has multiple etiologies—developmental, traumatic, neoplastic, inflammatory/infiltrative, vascular, infective. Causes of pituitary insufficiency include pituitary adenomas or other intrasellar and parasellar tumors, inflammatory and infectious destruction, surgical removal, radiation-induced destruction of pituitary tissue, traumatic brain injury (TBI), subarachnoid hemorrhage, and postpartum pituitary necrosis (Sheehan syndrome). Similar diseases originating in the hypothalamus or pituitary stalk may also result in pituitary insufficiency. Prevalence of Hypopituitarism in Indian society is not well established, but is estimated to be 45.5 per 100000 in a Caucasian based study. Presenting features of hypopituitarism may be due to hypogonadism, hypothyroidism or hypogonadism and studies have shown LH/FSH deficiency to be the commonest mode of presentation. Presentation varies from asymptomatic to acute collapse, depending on the etiology, rapidity of onset, and predominant hormones involved. Initially, a patient with any hormone deficiency may be asymptomatic. Individuals with the following deficiencies present with the indicated condition:

- ACTH deficiency - Adrenal (cortisol) insufficiency
- TSH deficiency - Hypothyroidism
- Gonadotropin deficiency - Hypogonadism
- GH deficiency - Failure to thrive and short stature in children; most adults are asymptomatic, but some may experience fatigue and weakness and decreased quality of life
- ADH deficiency - Polyuria and polydipsia

Other presenting features may be attributable to the underlying cause. A patient with a space-occupying lesion may present with headaches, double-vision, or visual-field deficits. A patient with large lesions involving the hypothalamus may present with polydipsia/polyuria or, rarely, syndrome of inappropriate secretion of antidiuretic hormone (SIADH).

Selectivity of hormonal loss in hypopituitarism has been explained by varying resilience of individual pituitary cell lineages to insult. The order of diminished trophic hormone reserve is GH>FSH> LH>TSH> ACTH, thus reflecting an attempt by the body to preserve important hormones (ACTH) to the very end of the disease. Data regarding hypothyroidism as a manifestation of hypopituitarism is limited, but as demonstrated in our case, hormone replacement therapy is the mainstay of treatment and lead to complete resolution of symptoms.

Hormonal studies should be performed in pairs of target gland and their respective stimulatory pituitary hormone for proper interpretation, as follows:

- ACTH (Cortrosyn) stimulation test (or morning cortisol and ACTH)
- TSH and thyroxine
- FSH, LH, and either estradiol (if amenorrheic) or morning testosterone (as appropriate for sex)
- Prolactin
- GH provocative testing

Stable patients who are diagnosed with hypopituitarism have a favorable prognosis with replacement hormone therapy. Patients with acute decompensation are in critical condition and may have a high mortality rate.

Finally complete evaluation of hyponatremia is exceedingly important as demonstrated in our case—correction of the sodium levels without investigating the cause led to a delay in diagnosis in this case. One should have a high index of suspicion for hypopituitarism as it may be more common than was thought.

END NOTE

Author Information

1. Dr. Karthick Ganesh, Resident, General Medicine, AIMS, Kochi.
2. Dr. Baiju Faizal, Assistant Professor, Department of General Medicine, AIMS, Kochi.
3. Dr. M G K Pillai, Professor, Department of General Medicine, AIMS, Kochi. E-mail: kar1411.g@gmail.com

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