

## CORRESPONDENCE

## Letter to the Editor in Response to Article “Severe Hyponatremia as an Uncommon Presenting Feature of Pituitary Macroadenoma”

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Sir,

I came across an interesting case of severe hyponatremia as an uncommon presenting feature of pituitary macroadenoma published in JAPI August 2018. This was a case of a 60 years old gentleman who presented with vomiting, giddiness, blackout and fatigue. On examination patient was found to be hypotensive, tachycardia was present, he also had dry tongue and loss of skin turgor (all signs of hypovolemia). On investigating he was found to have hyponatremia with normal potassium, normal renal and liver function test. ABG showed respiratory and metabolic alkalosis. Investigating further the authors have reported low cortisol, aldosterone and testosterone with a low ACTH, and diagnosed it as secondary Addison's due to pituitary tumor as the cause of hyponatremia.

Although a very well investigated and written case, we have a few queries and comments

1. The patient had hyponatremia and all signs of hypovolemia.

His serum osmolality has been reported as 288.5 mosm/l whereas the calculated osmolality is 231 mosm/l. If the reported osmolality is measured then the reason for this osmolar gap is not clear.

2. With this grade of hyponatremia and low osmolality, normal response of the kidney would be to produce a highly dilute urine with urine osmolality of < 100 mosm/l, but the authors have reported a urine osmolality of 373 mosm/l which is inappropriately high for this patient and indicate a state of excess of ADH. So this patient has a syndrome of excess of ADH. This can very well be explained in this patient due to the following reasons:

- a. presence of hypovolemia causes increase ADH release (normal osmotic response),
- b. vomiting is a strong non-osmotic stimuli for ADH release,
- c. presence of pituitary macroadenoma causes decrease ACTH which causes decrease cortisol release. Decrease cortisol release is a stimulus for release of ADH

Furthermore this patient is having as high urinary loss of sodium which is because of presence of excess of Atrial natriuretic peptide (ANP) in SIADH. The estimation of urinary sodium was done over a period of 24 hours during which time the patient was on saline

infusion and salt replacement, and it is well known that in SIADH the urinary sodium excretion depends upon the salt intake of the patient. So this patient probably had an underlying SIADH, and developed vomiting which can explain the presence of hypovolemia in him.

3. The authors have reported that the aldosterone levels were low in this patient but ACTH stimulation test done showed a normal adrenal reserve and a suppressed pituitary adrenal axis. The secretion of aldosterone is not dependent on pituitary adrenal axis instead it is dependent on volume status and renin angiotensin aldosterone axis. In a patient who is hypovolemic has increased renin secretion which causes increased aldosterone secretion when the adrenal gland is normal. So in this patient we expected a high aldosterone levels. This is further supported by the fact the serum potassium is normal and not high, the ABG is showing metabolic alkalosis. The authors have not reported TTKG values which would have further supported the diagnosis. The diagnosis of secondary Addison's as the cause of hyponatremia is not explainable.

So in our opinion this is a patient who developed symptomatic hyponatremia secondary to SIADH, which is secondary to pituitary macroadenoma and not due to secondary Addison's.