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A CASE REPORT ON SERUM CORTISOL INDUCED SIADH ASSOCIATED WITH SEVERE HYPONATREMIA

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ABSTRACT
Cortisol production is reduced in primary adrenal insufficiency. Electrolyte imbalance develops in patients, which can be severe and life-threatening. We discuss the example of a 55-year-old male who was taken to the hospital with chief complaints of vomiting since20 days. On evaluation the test results revealed chronic hyponatremia. The underlying reason was discovered to be primary adrenal insufficiency after further investigation. Our case study demonstrates that extreme hyponatremia can appear with neurological signs and symptoms. In the case of this patient, correction of hyponatremia, treatment with Tab. tolvaptan (Selective, competitive vasopressin receptor- 2 antagonist), Inj. ondansetron (Anti-emetic) and water restriction resulted in full clinical recovery.

KEYWORDS: Adrenal insufficiency, Cortisol, Hyponatremia,SIADH (Syndrome of inappropriate antidiuretic hormone secretion).

INTRODUCTION
Adrenal insufficiency (AI) is an illness in which adrenal cortisol production is either absent or insufficient. Direct adrenal insufficiency is the cause of primary AI (PAI), Secondary AI (SAI) is more common and is caused by disorders of the pituitary gland, whereas tertiary AI (TAI) is caused by diseases of the hypothalamus.[1] It can be life-threatening and its current prevalence is estimated to be between 100 to 140 instances per million, making it extremely rare.[2-3] Early detection of adrenal insufficiency is important to avert the potentially fatal consequences of severe hemodynamic and cardiovascular insufficiency.[4]

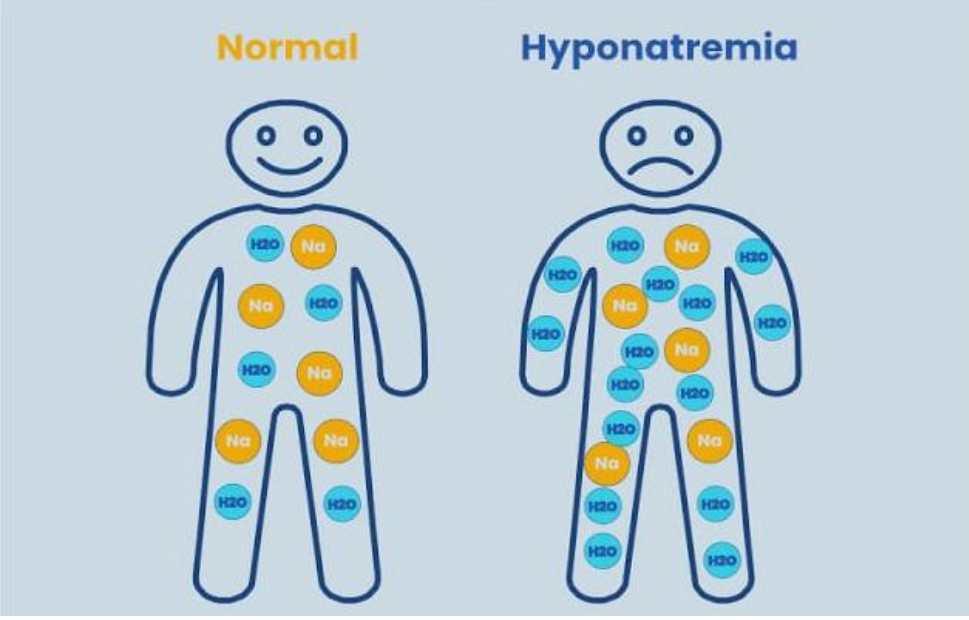

Figure: 1: Depiction of fluid and sodium content in hyponatremia.

CASE PRESENTATION

A 55-year-old male presented to the emergency department with complaints of recurrent episodes of vomiting associated with nausea and fatigue since past 20 days. Priorly the patient was taken to a local hospital where he was presented with vomiting and spinning sensation along with difficulty in walking. Post discharge patient developed hiccups which subsided after being presented here. His past medical history revealed that he has known complaints of HTN, DM and CAD. His last routine endoscopic health check-up revealed hiatus hernia, Grade-A oesophagitis. The patient's surgical history reveals that he underwent Coronary Artery Bypass Graft (CABG) 4 years back. MRI whole spine done outside showed severe compression at cervical region.

On admission, his BP was 130/80 mm/Hg, RR was 20 bpm and Temperature was 98.6 (F). The patient showed some neurological manifestations like loss of verbalization, altered behaviour and started giving starring looks. His systemic examination was unremarkable. His biochemistry reports resulted low serum sodium levels of 108 mmol/L, chloride of 90mmol/L, serum osmolality of 218 mOsm/kg, urine sodium of 220 mmol/L, serum uric acid of 1.3 mg/dL. The patient's cortisol levels were extremely low showing 9.84 ng/mL. His findings revealed consistent euvolemic hyponatremia. Additional tests were done to evaluate adrenal functions which resulted in elevated adrenocorticotropic hormone (ACTH) level (1201 pg/dl). To rule out any other secondary causes of hyponatremia relevant investigations such as chest X-ray, MRI brain plain and MRI cervical spine were done. The patient was haemodynamically stable and was discharged.

Table: 1: Laboratory values of the patient.

Labs	Patient's Results	Reference Range
Serum sodium	108 mmol/L	136 – 145 mmol/L
Chloride	90 mmol/L	98 – 107 mmol/L
Cortisol	9.84 ng/ml	54.94 – 287.56 ng/ml
ACTH	1201 pg/dl	9 – 52 pg/dl
Urine sodium	220 mmol/L	54 – 190 mmol/L
Serum osmolality	218 mOsm/kg	275 – 295 mOsm/kg
Serum uric acid	1.3 mg/dl	3.4 – 7.0 mg/dl

The patient was initially treated with hypertonic saline along with tab. Tolvaptan for the rectification of severe hyponatremia levels. On further treatment the patient's sodium level was corrected gradually to above 128 mmol/L, hypertonic saline was stopped. Further, the patient was restricted to fluids up to 1.5 litres to 2 litres per day. The correction of sodium levels for the next few days resulted in direct symptomatic improvement of the patient.

CASE DISCUSSION

A peptide hormone, corticotropin releasing hormone (CRH) stimulates both secretion and synthesis of ACTH from the anterior pituitary gland. Thus, when the pituitary gland does not make sufficient amount of ACTH leads to decreased production of cortisol resulting in cortisol deficiency. Ultimately Cortisol deficiency results in elevated hypothalamic secretions of CRH which is an ADH secretagogue leading to alteration in the levels of anti- diuretic hormone (ADH) eventually causing cortisol induced SIADH in association with hyponatremia.[5]

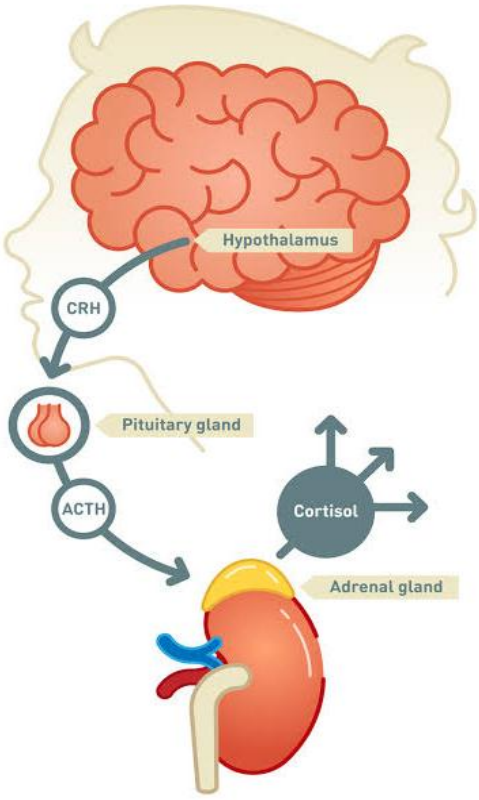

Figure 2: Adrenal gland and its functioning.

In our case, initial clinical examination and laboratory findings indicated the presence of

euvolemic hyponatremia. Our considerations for differential diagnoses included syndrome of inappropriate anti-diuretic hormone (SIADH), adrenal insufficiency and drug abuse. In this patient, the reports revealed there was significant elevation in ACTH levels (1201 pg/dl), low cortisol levels (9.84 ng/ml). Over a period of time lack of cortisol mediated negative feedback on ADH resulted in severe hyponatremia (108 mmol/L). As compared to acute cases of hyponatremia the chronicity of adrenal insufficiency in this patient, resulted in more severe clinical manifestations. Markedly elevated ACTH levels as well as low cortisol levels determined adrenal sufficiency in this patient. Further investigations in the patient revealed decreased urine sodium (220 mmol/L), decreased serum osmolality (218 mOsm/ kg). The patient was found to be euvolemic on evaluation. The decreased serum osmolality, increased urine sodium, decreased plasma sodium, decreased cortisol levels, euvolemic condition were compatible with SIADH, probably secondary to adrenal insufficiency in this patient.

Therapeutic approach for adrenal insufficiency targets on replenishing the hormone deficiency. The patient was treated with tab. Tolvaptan and was kept on fluid restriction of 1.5 litres to 2 litres of fluids per day. The patient showed symptomatic improvement with complete rectification of hyponatremia and was discharged with instruction to follow up regularly.

CONCLUSION

SIADH is one of the rarely encountered clinical illness. However, our case report signifies that SIADH in association with chronic hyponatremia can occur with declining cortisol and serum sodium levels as low as 9.8ng/ml and 108mmol/L respectively could absolutely have resulted in neurological manifestations. There are several confounding factors for chronic hyponatremia, as in this case caused due to adrenal insufficiency and SIADH. Therefore, a definitive lab interpretation must be done to determine the predisposing factors and the underlying cause of hyponatremia.

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