CONCLUSION
Although triphasic phase of central DI is relatively rare, it is important to identify the phase, as the treatment differs depending on the phase.

EP_A111

UNRAVELING THE ENIGMA: TRIMETHOPRIM-SULFAMETHOXAZOLE-INDUCED SIADH

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Seetha Devi Subramanian, Gerard Jason Mathews, Joel Mathews, Teh When Yee, Noor Rafhati Adyani Abdullah, Shartiyah Ismail, Nor Shaffinaz Yusoff Azmi Merican
Endocrinology Unit, Department of Medicine, Hospital Sultanah Bahiyah, Malaysia

INTRODUCTION/BACKGROUND
Syndrome of inappropriate antidiuretic hormone secretion (SIADH) presents a complex clinical scenario characterized by the aberrant secretion of antidiuretic hormone (ADH), leading to hyponatremia, water retention and potential neurological manifestations. Trimethoprim-sulfamethoxazole (TMP-SMX, Bactrim) is a potential cause of medication-induced SIADH.

CASE
A 21-year-old female came in with left gluteal abscess and newly diagnosed diabetes mellitus complicated by diabetic ketoacidosis (DKA). Following treatment for DKA and abscess incision and drainage, which grew Staphylococcus argenteus, she received intravenous cloxacillin for 7 days. Antibiotics were then shifted to oral TMP-SMX as she was deemed fit for discharge. After 3 days on TMP-SMX, she developed severe hyponatremia with a sodium level of 114 mmol/L, despite having baseline sodium levels ranging between 135-143 mmol/L. Despite hydration with 4 L of NaCl per day, her serum sodium levels continued to decline, reaching a nadir of 108 mmol/L. She was then referred to the medical team for further management.

Urine sodium and osmolarity were elevated at 95 mEq/L and 316 mOsm/L, respectively, with a low serum osmolality at 262 mOsm/L. Morning cortisol level and thyroid function tests were within normal level and she was euvolemic. A diagnosis of medication TMP-SMX-induced SIADH was made. She was started on fluid restriction of less than 1 L per day. Serum sodium levels gradually improved to 130 mmol/L, with stable electrolytes, and renal function and she was discharged well.

CONCLUSION
TMP-SMX is a potential cause of medication-induced SIADH. Additionally, trimethoprim (TMP) shares structural similarities with amiloride and functions on the identical epithelial sodium channels (eNAC) in the distal nephron, leading to natriuresis and hyponatremia. Prompt identification of the cause of hyponatremia (diuresis vs SIADH) is crucial in averting severe complications linked with hyponatremia.

EP_A112

WHEN TWO DIABETES MET: HYPERGLYCAEMIC EMERGENCY OR VASOPRESSIN DISORDER?

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Afifah Kamarudin, Nurul Thohirah Ahmad, Jen Hoong Oon, Nor Afidah Karim
Department of Internal Medicine, Hospital Tuanku Ja'afar Seremban, Malaysia

INTRODUCTION/BACKGROUND
Central diabetes insipidus (CDI) is caused by decreased secretion of or resistance to ADH. The clinical and laboratory findings may be similar to the hyperosmolar hyperglycaemic state (HHS). We reviewed case notes, investigation results, imaging and treatment options based on literature review.

CASE
A 30-year-old female with history of gestational diabetes mellitus presented with 3-day history of vomiting and fever. She also had polyuria, polydipsia and fatigue for the past 2 years. Her blood sugar level was 24.9 mmol/L, serum osmolarity was 346 mOsm/L and serum sodium was 162 mmol/L, with no acidosis or ketosis. The patient was diagnosed with HHS and received appropriate treatment. However, she continued to experience polyuria. Further investigation revealed weight gain, irregular menstrual cycles and recent absence of menstruation. Subsequent investigations revealed features of diabetes insipidus (DI) (serum sodium: 160 mmol/L, serum osmolality: 350 mOsm/kg, urine osmolality: 114 mOsm/kg). Following the administration of desmopressin, the urine osmolality increased to 505 mOsm/kg. Additional tests conducted showed normal prolactin, cortisol and thyroid function, but low IGF-1 and hypogonadotropic hypogonadism. The patient was started on regular sublingual desmopressin and her symptoms improved. She is currently awaiting an MRI of the pituitary gland.