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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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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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.

The patient's initial presentation resulted in treatment for HHS. However, subsequent investigation uncovered the presence of CDI, which has been obscured by the diabetes mellitus. In younger patients who present with CDI, hypophysitis is typically the cause, reported to occur in up to 50% of patients. \Treatment decisions should be guided by clinical evaluation and imaging, as patients with pituitary dysfunction but no mass effect and likely lymphocytic hypophysitis may be managed with medical therapy and close monitoring.

CONCLUSION

Clinicians should have a high index of suspicion of CDI among patients manifesting with possible HHS who do not improve despite adequate control of hyperglycemia.

EP_A113

LOCAL EXPERIENCE WITH TARGETED RADIONUCLIDE THERAPY IN MALIGNANT PHEOCHROMOCYTOMA AND PARAGANGLIOMA

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INTRODUCTION/BACKGROUND

Targeted radionuclide therapy (TRT) is a promising therapeutic option for patients with malignant pheochromocytoma and paraganglioma (PPGL) but it is not widely available locally.

METHODOLOGY

We conducted a retrospective cohort study of patients with malignant PPGL from 2000 to 2023.

RESULTS

We report the experience of TRT among 15 patients with malignant PPGL (26% pheochromocytoma, 60% paraganglioma, 13% combined) under follow-up in a tertiary endocrine referral centre from 2000 to 2023. There was equal gender distribution with a median of 41 years at diagnosis. They had elevated 24-hr urine normetanephrine (100%), 3-methoxytyramine (53.3%) or urine metanephrine (27%). A total of 11 patients had multiple operations with residual primary and metastatic tumours, 2 had recurrence after initial complete resection and another 2 had unresectable primary tumour. The choice of TRT was based on avidity in functional imaging and consensus from multidisciplinary meetings. Ten patients had peptide receptor radionuclide therapy (PRRT) and 5 patients had iodine 131-meto-iodo-benzyl-guanidine (MIBG). A 177Lu-DOTATATE was used for PRRT with a mean dose of 201.23 mCi (7.47GBq/cycle). There was a reduction in both urine normetanephrine (93%) and requirements for antihypertensive medications (80%) after TRT. Using Response Evaluation Criteria in Solid Tumours (RECIST), disease control rate was 40% after 4 cycles of PRRT (n = 4) or MIBG (n = 2). Among patients with disease progression, a subsequent plan was additional TRT cycles up to a total of 6 cycles (n=3), chemotherapy (n=2), or watchful waiting (n = 1). One patient with SDHB mutation, who had multimodal therapies including multiple surgeries, chemoembolization, PRRT and chemotherapy with temozolamide, succumbed to her progressive disease 20 years after diagnosis. With regards to toxicities using Common Terminology Criteria for Adverse Events (CTCAE), there were grade I hypotension post PRRT (n = 1), grade I leucopenia (n = 1) and grade I-II renal impairment (n = 3).

CONCLUSION

TRT is well tolerated and worthy of extensive research to explore full potential in the treatment of advanced or non-resectable PPGL.

EP_A114

HYPERNATREMIC DEHYDRATION AND ACUTE MASSIVE PULMONARY EMBOLISM: COINCIDENCE OR TRUE RISK FACTOR

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INTRODUCTION/BACKGROUND

Diabetes Insipidus (DI) is a common complication after extensive transcranial surgery for craniopharyngioma. DI mainly causes impairment of the sense of thirst and vasopressin (AVP) secretion. This puts the patient at risk of severe dehydration and hypernatremia. Venous thromboembolism is one of the potentially fatal complications which can occur due to severe dehydration and hypernatremia.

CASE

We report a 22-year-old Malay male, college student, with underlying panhypopituitarism and chronic diabetes insipidus post-removal of craniopharyngioma at the age of 8 years with acute massive bilateral pulmonary embolism (PE). He presented to our Emergency Department with acute onset of shortness of breath and chest pain for a day. Although feeling unwell, he was still able to ambulate and attend school.