EP_A109

OBSTACLES IN MANAGING GIANT PROLACTINOMA: A SUDDEN RESURGENCE WITH NEW ONSET SEIZURE IN GIANT PROLACTINOMA

https://doi.org/10.15605/jafes.039.S1.120

Siti Zakiah Ahmad Daud, Syarifah Syahirah Syed Abas, Nor Afidah Karim, Yong Lit Sin, Noor Lita Adam

Endocrine Unit, Department of Medicine, Hospital Tuanku Ja'afar Seremban, Malaysia

INTRODUCTION/BACKGROUND

Prolactinoma is the most common type of secretory pituitary adenoma, caused by oversecretion of prolactin (PRL). Giant prolactinomas are uncommon, accounting for only 2-3% of all prolactinomas and are more common in males. Management of giant prolactinomas is also more challenging as these patients require a higher dose of dopamine agonist (DA) and are at risk of developing aggressive prolactinomas or carcinoma.

CASE

This is a case of a 29-year-old male who was diagnosed with giant prolactinoma at the age of 24, with extension to both cavernous sinuses (knops 4), causing 3rd ventricle compression. He was treated medically with an increasing dose of DA for the last 5 years. There was an improvement in both prolactin level and tumour size with cabergoline 1.5 mg daily (total weekly dose 10.5 mg) until the current presentation when he had a sudden increase in prolactin levels accompanied by new onset seizures.

Assay interference was excluded after 3 samples of serum prolactin levels sent to different platforms revealed almost similar results. Macroprolactinemia was also excluded after the PEG test came back negative. The cranial MRI also revealed a minimal increase in tumour size without any indication of tumour aggressiveness. Upon further inquiry, he admitted being less compliant with his cabergoline dose for the past year, with no apparent reason. He was reminded to strictly adhere to the prescribed dosage of DA, and the follow-up MRI of the pituitary was planned after 6 months. Indications for adjunctive therapy (i.e., transsphenoidal surgery for tumour enlargement despite compliance with medication) were also explained to the patient.

CONCLUSION

This case highlighted the challenges in managing giant prolactinomas, the differential diagnoses of biochemical relapse with new-onset seizure and the importance of medication adherence. We also highlight the indication for surgery in macroprolactinoma.

EP_A110

TRIPHASIC PHASE OF CENTRAL DIABETES INSIPIDUS (DI) POST TRANSPHENOIDAL SURGERY: A NORTHERN REGION GOVERNMENT HOSPITAL EXPERIENCE

https://doi.org/10.15605/jafes.039.S1.121

Tat Chee Calvin Chang, Gerard Jason Mathews, Nor Shaffinaz Binti Yusoff Azmi Merican

Endocrinology Unit, Department of Medicine, Hospital Sultanah Bahiyah

INTRODUCTION/BACKGROUND

The transsphenoidal pituitary surgery approach is a potential cause of central diabetes insipidus (DI) due to its impact on the pituitary stalk. Triphasic DI is a rare manifestation of central DI. In the initial phase, patients experience polyuria secondary to axonal shock or injury of vasopressin-secreting neurons in the hypothalamus. In the second phase, there is transient inappropriate antidiuretic hormone secretion (SIADH) due to vasopressin leakage from the damaged posterior pituitary tissues. The third phase ensues if >80% of vasopressin-secreting neurons are destroyed.

CASE

A 44-year-old Malay female presented with sudden slurring of speech, numbness of the right upper limb and headaches. Her cranial CT and MRI showed pituitary macroadenoma (1.1 x 1.5 x 1.1 cm). Pituitary hormone profile was normal. Ten months later, she developed bitemporal hemianopsia with a repeated cranial MRI showing unchanged size of the sellar lesion. Eventually, she underwent transsphenoidal surgery and tumour excision for pituitary macroadenoma.

About 8 hours post-op, she developed polyuria with urine output of 400 ml/hour, sodium level: 145 mmol/L, urine sodium: <20 mmol/L, serum osmolality: 299 mmol/L, urine osmolality: 110 mmol/L which was consistent with central DI. Subcutaneous desmopressin was given on days 1 to 3 post-op.

On day 7 post-op, she developed SIADH as evidenced by serum osmolality of 241 mmol/L, urine osmolality of 527 mmol/L and urine sodium of 124 mmol/L.

Upon entering day 11, she had another episode of polyuria (200 ml/hour). Laboratory tests revealed serum sodium: 126 mmol/L, serum osmolality: 269 mmol/L, urine osmolality: 111 mmol/L and urine sodium: 111 mmol/L which were suggestive of central DI in the triphasic phase. Hence, subcutaneous desmopressin was resumed. Sodium levels normalized and she was discharged home clinically well on sublingual desmopressin.

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

https://doi.org/10.15605/jafes.039.S1.122

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?

https://doi.org/10.15605/jafes.039.S1.123

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.