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Acute Severe Hyponatraemia in a Patient with Right Eye Ptosis

https://doi.org/10.15605/jafes.034.S65

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INTRODUCTION

Hyponatraemia is a common electrolyte imbalance in oncology. Syndrome of inappropriate anti-diuretic hormone secretion (SIADH) contributes to approximately 30% of cases. However, it remains a diagnosis of exclusion. In oncology, other possible contributing causes include reduced sodium intake, gastrointestinal and renal losses, chemotherapy and radiotherapy. However, for hyponatraemia in head and neck tumours, pituitary or hypothalamic involvement needs to be ruled out. We report a case of severe hyponatraemia and complex ophthalmoplegia in a patient with nasopharyngeal carcinoma (NPC) as a presentation of pituitary extension.

The clinical records of a patient who was diagnosed with NPC were reviewed.

CASE

A 43-year-old gentleman presented with double vision, headache and weight loss for 2 months. It was associated with right third and sixth cranial nerve palsies, as well as left submandibular lymphadenopathy. Computerized tomography scan and lymph node biopsy confirmed the diagnosis of nasopharyngeal carcinoma. Prior to commencement of chemotherapy, he also presented with acute severe hyponatraemia which was resistant to supportive treatment. Urine osmolality and serum osmolality pointed toward SIADH, but the patient was not responsive to fluid restriction and hypertonic saline correction. Pituitary gland function tests and magnetic resonance imaging subsequently confirmed pituitary extension of the NPC causing central hypocortisolaemia with concomitant SIADH. Hyponatraemia was successfully treated with oral hydrocortisone and oral sodium chloride.

CONCLUSION

This case study illustrates important presentations of NPC progression. Complex ophthalmoplegia and hyponatraemia are both warning signs of intracranial NPC extension.

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A Rare Case of Double Adrenocorticotropic Hormone-Secreting Pituitary Adenoma

https://doi.org/10.15605/jafes.034.S66

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INTRODUCTION

When distinct pituitary hypersecretory manifestations coexist, the differential diagnoses include plurihormonal or multiple pituitary adenomas. We describe a rare case of Cushing's disease and central diabetes insipidus caused by two non-contiguous pituitary adenomas, one located within the anterior pituitary and the other in the infundibulum.

CASE

A 14-year-old female presented with unexplained weight gain, central obesity, hirsutism, polyuria and polydipsia. Hormonal studies indicated Cushing's disease, and magnetic resonance imaging showed a small focal area of delayed enhancement in the right pituitary gland. While other anterior pituitary hormonal tests were negative, osmolality and water deprivation test confirmed central diabetes insipidus. Endoscopic transsphenoidal surgery revealed 2 lesions containing cheesy-like material at the posterior part of the anterior pituitary and another compressing the infundibulum. The 2 clearly separated pituitary adenomas identified in the same gland were completely resected. Immunohistochemistry and pathology revealed that the double adenomas were positive for adrenocorticotropic hormone (ACTH), thyroid stimulating hormone, growth hormone, luteinising hormone, prolactin and follicle stimulating hormone. Postoperatively, the levels of ACTH and cortisol decreased rapidly. However, she developed panhypopituitarism with persistent diabetes insipidus, requiring hormonal replacement therapy.

Multiple pituitary adenomas (MPA) are defined as 2 or more immunocytochemically and/or morphologically distinct tumours that are detected in only 0.37 to 2.6% of surgical specimens and in 1.6 to 3.3% of Cushing's disease patients. Our patient is the youngest reported patient to our knowledge so far and is considerably rare, due to the presence of a second pituitary adenoma in the same gland detected only intraoperatively and not radiologically, isolated ACTH-secreting tumours, and clinical presentation of diabetes insipidus.

CONCLUSION
The coexistence of double adenomas can pose diagnostic and management challenges for the pituitary neuroendocrine team and is a common cause for surgical failure. Intraoperative evaluation is important in the identification of multiple pituitary adenomas in a patient presenting with multiple secretory manifestations.

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Brittle Bones and Leaking Phosphate

https://doi.org/10.15605/jafes.034.S67

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INTRODUCTION
Fanconi syndrome is an established cause for low bone mineral density (BMD). Confirmed cases of acquired Fanconi syndrome due to tenofovir have been reported worldwide. The mean duration of therapy with tenofovir before the development of Fanconi syndrome is reported to be 11 months. The implicated agent was discontinued in all cases after which renal function tests and electrolytes normalised. We present a case of acquired Fanconi syndrome following tenofovir use.

CASE
A 57-year-old male with Hepatitis B infection who had been on tenofovir for 5 years presented with a low impact calcaneal fracture while standing up from a squatting position 3 years ago. Over the past two years, he experienced gradually worsening proximal muscle weakness and weight loss of 6 kg. Physical examination revealed a man of small build with proximal muscle weakness and tenderness. Blood parameters showed hypokalaemia and hypophosphatemia with inappropriately elevated urinary phosphate and potassium clearance. Thyroid function and serum testosterone were normal. His 25-hydroxyvitamin D levels were sufficient. Electromyography reported diffuse neurogenic pattern with secondary myogenic changes suggestive of a metabolic cause. Abdominal ultrasound revealed bilateral renal calculi. Dual x-ray absorptiometry scan showed osteoporosis at the lumbar spine and distal third of the radius. A diagnosis of acquired Fanconi syndrome associated with tenofovir therapy was made. He was started on oral phosphate and potassium supplements while tenofovir was replaced with entecavir. Upon review 6 months later, he was much better with no muscle pain or weakness. Repeated serum potassium and phosphate levels were within normal limits.

CONCLUSION
Tenofovir use is associated with acquired Fanconi syndrome which can lead to osteoporosis. A high index of suspicion is necessary among patients on this medication who present with low impact fractures as timely intervention can prevent significant morbidity.

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The Invisible Evil Twin of an Adrenal Adenoma

https://doi.org/10.15605/jafes.034.S68

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INTRODUCTION
Primary aldosteronism (PA) causes a persistently elevated blood pressure (BP) due to excessive release of the hormone aldosterone from the adrenal glands. It can be cured with surgical resection of the aldosterone-secreting adenoma leading to resolution of hypertension and reduction in cardiovascular risk. There is known discordance between identification of adenoma with computed tomography (CT) and confirmation of aldosterone hypersecretion with adrenal venous sampling (AVS).

CASE
We present the case of a man with previous ischemic heart disease who presented with resistant hypertension. He had been diagnosed with essential hypertension 5 years prior. Investigations for secondary causes of hypertension were performed, as he subsequently required 5 anti-hypertensive medications to control his hypertension. Work-up revealed an elevated serum aldosterone of 924 pmol/L [normal range (NR) 111 to 860] with suppressed plasma renin activity of 0.4 ng/mL/hr (NR 1.5 to 5.7); and aldosterone-to-renin ratio of 2060 (NR <750). Saline suppression test confirmed the diagnosis, with failure of suppression of aldosterone with salt loading. CT of the adrenal glands revealed a left adrenal adenoma measuring 1.4 cm x 1.5 cm with a Hounsfield Unit (HU) of 12 and absolute washout of 60%. The right adrenal gland was normal.

AVS was performed. There was lateralisation to the right adrenal gland indicating aldosterone hypersecretion but with normal adrenal imaging. The Lateralisation index ratio was 8.6 (NR <3). The patient subsequently underwent a repeat AVS which produced similar results. One month later, he underwent laparoscopic right adrenalectomy which improved his BP control. Histologic features were consistent with adrenal cortical adenoma.

CONCLUSION
This case highlights the importance of recognizing the need to investigate for secondary causes of hypertension. It also underscores the importance of dynamic tests such as AVS to confirm hypersecretion of aldosterone from the correct adrenal gland resulting in the best treatment option.