Adult E-Poster

EP_A003

HYPOKALEMIA-INDUCED NEPHROGENIC DIABETES INSIPIDUS IN REFEEDING SYNDROME

https://doi.org/10.15605/jafes.040.S1.011

Wei Ton Wong, Jia Chuan Chong, Raja Nurul Azafirah Raja Amir Shah
Department of Internal Medicine, Hospital Sultanah Nur Zahirah, Malaysia

INTRODUCTION/BACKGROUND

Nephrogenic diabetes insipidus is a rare disorder in which the body produces excessive amounts of urine. It can be caused by a genetic mutation or acquired factors such as certain medications (lithium, amphoterin-B), electrolyte imbalance (hypokalemia, hypercalcemia), chronic kidney disease or obstructive uropathy. In this case report, we describe a case of refeeding syndrome followed by hypokalemia-induced nephrogenic diabetes insipidus.

CASE

A cachectic 37-year-old male with BMI of 15.0 kg/m^2 with underlying mild intellectual disability and history of pulmonary tuberculosis (TB) who had completed his TB treatment presented at the emergency department with 2 weeks history of vomiting and 3 weeks history of bilateral lower limb weakness, loss of appetite and weight loss. Vital signs were stable. Systemic examination revealed bilateral lower limb weakness with power of 4/5. Laboratory data were significant for hypokalemia (1.8 mmol/L), hypophosphatemia (0.48 mmol/L) and hypocalcemia (1.6 mmol/L). The patient was admitted for electrolyte correction. At the ward, patient was noted to be producing excessive amounts of urine ranging from 4 to 10 L/day. Water deprivation test was performed. It showed failure to increase urine osmolality with water deprivation and lack of response to desmopressin suggestive of nephrogenic diabetes insipidus. Spot urine potassium was low at 9.8 mmol/L. With multiple corrections of electrolytes, hypokalemia, hypophosphatemia and hypocalcemia gradually resolved. The patient was started on a low-calorie, high-protein diet for the consideration of refeeding syndrome. Following correction of electrolytes with potassium reaching a stable level of 4 mmol/L, on the 17th day of admission, the daily urine output was restored to a normal volume of approximately 2 L/day. After three days, he was discharged.

CONCLUSION

Patients with polyuria and hypokalemia should be evaluated for nephrogenic diabetes insipidus. Hypokalemia-induced nephrogenic diabetes insipidus can be reversed by correcting hypokalemia.

EP_A004

A CASE OF SEVERE HYPERCALCAEMIA SECONDARY TO PARATHYROID CARCINOMA

https://doi.org/10.15605/jafes.040.S1.012

Abdullah Shamshir Abd Mokti, Meng Loong Mok, Shireene Ratna Vethakkan
Endocrine Unit, University Malaya Medical Centre, Malaysia

INTRODUCTION/BACKGROUND

Parathyroid carcinoma is a rare malignancy, accounting for only 0.005% of all cancers and about 0.5-1% of parathyroid disorders with similar incidence in male and females. It usually presents with manifestations of severe hypercalcaemia with bone involvement and nephrolithiasis, associated with markedly raised parathyroid hormone; although presentations can be variable.

CASE

We report a case of a 44-year-old Chinese male with underlying hypertension, chronic kidney disease stage 3A (eGFR 52 mL/min/1.73 m^2) who was incidentally found to have PTH–dependent severe hypercalcaemia (corrected calcium 4.56 mmol/L), hypophosphataemia with iPTH 102.1 (>7 times the upper limit of normal) during admission for left cheek subcutaneous abscess. Hypercalcaemia was managed with calcitonin, bisphosphonate alongside aggressive saline diuresis resulting to a serum calcium of 2.85 mmol/L. Ultrasound of the neck showed a well-defined ovoid hypoechoic lesion, caudal to the lower pole of the left thyroid lobe measuring 1.4 x 1.5 x 2.0 cm consistent with left parathyroid adenoma, which was confirmed with parathyroid Sestamibi scintigraphy. Ultrasound of the kidneys revealed no nephrolithiasis while bone densitometry showed severe osteoporosis at the distal third of forearm and left neck of femur with T score -4.1 and 3.0 respectively. Surgical excision of the left parathyroid with intraoperative PTH monitoring was done with subsequent removal of the lesion leading to normalization of the PTH level. Histopathology examination revealed lymphovascular permeation with irregular nodular proliferation of parathyroid cells with transgressed boundaries which are features compatible with parathyroid carcinoma. Post-operatively, the patient was normocalcaemic but declined further surgical tumour clearance.

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CONCLUSION
This case highlights the need for a high index of clinical suspicion for the presence of parathyroid carcinoma pre-operatively in patients who exhibit severe hypercalcemia, markedly raised PTH and bone manifestations so that en bloc-resection of the parathyroid with ipsilateral partial thyroidectomy and central node dissection can be planned prior to surgery.

EP_A005

THYMIC HYPERPLASIA IN GRAVES' DISEASE: A DIAGNOSTIC AND MANAGEMENT CHALLENGE

https://doi.org/10.15605/jafes.040.S1.013

Lim Guat Yee[1] and Kuan Yueh Chien[2]
[1]Hospital Limbang, Sarawak, Malaysia
[2]Hospital Miri, Sarawak, Malaysia

INTRODUCTION/BACKGROUND
Thymic hyperplasia is a recognized but frequently underappreciated entity associated with Graves' disease (GD). It is often misinterpreted as a mediastinal mass, potentially leading to unwarranted biopsies or surgical intervention. The underlying pathophysiological mechanisms remain poorly understood. Spontaneous regression of the mediastinal mass following euthyroidism with effective thyrotoxicosis treatment supports a benign etiology. Here, we present a case of a young female with GD and an incidentally discovered anterior mediastinal mass, highlighting the diagnostic complexities that necessitated a multidisciplinary approach.

CASE
A 21-year-old female presenting with a large goiter, a thyrotoxic state (FT4 >100 pmol/L, TSH 0.01 mU/ml and anti-TSH receptor Ab >40 IU/L) with no thyroid ophthalmopathy was diagnosed with GD. Despite medical management, adequate control of her thyroid hormone levels proved to be challenging, prompting a surgical consultation for a potential thyroidectomy. To assess the extent of the goiter, computed tomography (CT) imaging was performed, revealing a grossly enlarged thyroid gland with mild tracheal narrowing and a well-defined, solid, enhancing 5.6 cm × 6.4 cm × 4.3 cm anterior mediastinal mass.

Given the initial concern for an ectopic thyroid gland or malignancy, performing an invasive biopsy was considered. However, a multidisciplinary team consisting of experts from endocrinology, surgery, respiratory medicine, radiology, and nuclear medicine reviewed the findings and concluded that the mass was most consistent with thymic hyperplasia. Considering the high surgical risk, a conservative approach was pursued, with the patient undergoing radioiodine therapy for thyrotoxicosis and serial imaging to monitor the mediastinal mass. Long-term outcomes are yet to be seen.

CONCLUSION
This case underscores the diagnostic challenges posed by thymic hyperplasia in patients with GD and the potential for misdiagnosis as a mediastinal pathology. Awareness of this association is crucial in order to avoid unnecessary surgical interventions. A multidisciplinary approach is essential for accurate diagnosis and optimal management, promoting a conservative therapeutic strategy when appropriate.

EP_A006

GRANULOMATOUS DISEASE-INDUCED SEVERE HYPERCALCEMIA

https://doi.org/10.15605/jafes.040.S1.014

Nur Farrah Anima Muhammad,[1] Fadzliana Hanum Jalal,[2] Mohd Khairul Mohd Kamil[3]
[1]Department of Internal Medicine Hospital Shah Alam, Selangor, Malaysia
[2]Endocrine Unit Hospital Shah Alam, Selangor, Malaysia
[3]Nephrology Unit Hospital Shah Alam, Selangor, Malaysia

INTRODUCTION/BACKGROUND
Hypercalcemia is commonly seen in granulomatous disease especially in sarcoidosis in around 40-50% cases; however, lower rates of association have been reported in tuberculosis. The etiology is due to the production of extrarenal 1-alpha-hydroxylase enzymes by activated macrophages seen in the granulomas. This will then lead to inappropriately elevated 1,25-dihydroxyvitamin D causing dysregulation of calcium metabolism.

CASE
A patient with a known case of disseminated tuberculosis (TB) was admitted to critical care with an initial impression of cerebral toxoplasmosis. Throughout his admission, blood parameters were closely monitored which revealed moderate to severe hypercalcemia ranging from 2.8-4.0 mmol/L with clinical features of nephrogenic diabetes insipidus (polyuria of 5440 ml urine output per day, hypernatremia ranging 147-157 mmol/L (135-145 mmol/L) and low urine osmolality 143 mOsm/kg). However, despite treatment with hydration, severe hypercalcemia resulted in the atypical presence of J-wave or Osborn wave on electrocardiogram (ECG). Hypothermia has been ruled out as his body temperature ranges from 36.7-37 °C. There is no interruption in his TB medications and iatrogenic