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.