CONCLUSION

Most patients responded well to 10 mg of empagliflozin and achieved sustained HbA1c at 6 months of treatment. However, a third of patients did not respond well to empagliflozin 10 mg, even after up-titrating to 25 mg. These finding suggests that if patients do not achieve at least 0.5% reduction in HbA1c with 10 mg dose, further significant reduction in HbA1c is unlikely to be achieved with up-titration to 25 mg for the next 3 months.

PA-A-36

CEREBELLAR ATAXIA ASSOCIATED WITH ANTI-GLUTAMIC ACID DECARBOXYLASE ANTIBODIES: A CASE REPORT

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Saraswathy Apparow[1] and Cheah Cheng Foong[2]
[1]Endocrine Unit, Specialized Diagnostic Centre, Institute for Medical Research, National Institute of Health, Kuala Lumpur, Malaysia
[2]Medical Department, Hospital Kapit, Sarawak, Malaysia

INTRODUCTION

Anti-glutamic acid decarboxylase (anti-GAD) - related cerebellar ataxia is the second most common cause of GAD antibody (Ab) spectrum disorders. It is characterised by cerebellar symptoms with elevated GAD Ab levels in the serum and cerebrospinal fluid (CSF). It commonly affects females associated with Type 1 DM or polyendocrinopathy. IVIG is the most effective immunomodulatory therapy.

CASE

We report a 34-year-old male diagnosed with Type 1 DM with high titer of serum anti-GAD Ab who first presented with cerebellar syndrome at the age of 12. At 15 years of age, HbA1c was 12% hence, insulin treatment was initiated. Initial diagnosis of neurodegenerative disorder was made in view of brain MRI findings showing cerebellar atrophy and family history of consanguineous marriage.

Laboratory investigation revealed high serum anti-GAD Ab titre >250 IU/ml. He was on basal-bolus insulin regimen and self-monitoring of blood glucose showed good control. There was no target organ damage. Furthermore, there was no progressive worsening of the neurological deficit. Repeated cranial MRI showed stable symmetrical hyperintensity in the atrophic middle cerebellar peduncles and pons with cerebellar atrophy. A lumbar puncture was performed and CSF analysis for anti-GAD Ab revealed remarkably high titre >250 IU/ml. Work-up for other causes of cerebellar ataxia and neurodegenerative disorders were negative. Immunomodulatory treatment was not initiated in view of non-progressive symptoms.

CONCLUSION

The unique association of autoantibody-mediated cerebellar ataxia and T1DM in this male patient is interestingly rare with childhood cerebellar syndrome as initial presentation before the diagnosis of Type 1 DM. Immunomodulatory treatment may be effective. We emphasize the importance of long-term follow-up, given the possibility of late development of other anti-GAD related neurological disorders and autoimmune polyendocrinopathy.

PA-A-37

T3 THYROTOXICOSIS SECONDARY TO GRAVES' DISEASE EXHIBITING RESISTANCE TO RADIOACTIVE IODINE-131 THERAPY

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Aimi Fadilah Mohamad, Fatimah Zaherah Mohamed Shah, Nur Aisyah Zainordin, Nur'aini Eddy Warman, Sharifah Faradila Wan Muhammad Hatta, Mohd Hazriq Awang, Rohana Abdul Ghani
Universiti Teknologi MARA (UiTM), Sungai Buloh, Malaysia

INTRODUCTION

Radioactive Iodine (RAI) therapy with Iodine-131 is commonly used as definitive therapy for Graves' Disease. It is especially useful when there is poor response to anti-thyroid medications. The failure rate for RAI therapy is approximately 15% and known predictors for failure are RAI doses of <13 mCi and prior methimazole therapy. Initial free T3 (fT3) and T4 (fT4) levels at presentation may also predict response to RAI therapy.

CASE

We present a case of a 44-year-old female with Graves' Disease and persistently elevated fT3 levels. Her main symptoms were weight loss, palpitations and severe panic and anxiety attacks. She had mild ophthalmopathy and a moderate goitre but no compression symptoms. She was treated with carbimazole for 2 years but was unable to achieve euthyroidism.

Her initial thyroid function tests showed TSH <0.01 mIu/L (NR: 0.27 – 4.2), fT4 >100 pmol/L (NR: 12 - 22) and fT3 >50 pmol/L (NR: 3.5 - 6.5). Thyroid peroxidase (TPO) antibodies were elevated at 692 IU/ml (NR <35). With carbimazole, her fT4 normalized (range: 13 - 19) but fT3 remained elevated (range: 8 - 13). Carbimazole dose was increased and fT3 normalized to 5.1 pmol/L but fT4 decreased to 1.7 pmol/L. Her TSH remained suppressed throughout. She received RAI at 20 mCi with immediate relapse after 4 weeks (fT4 >100). Eight months later, she had second RAI with 20 mCi but remained hyperthyroid within 6 months of follow-up.

CONCLUSION

Despite a total RAI dose of 40MCi, her Graves' Disease remained active and thyroidectomy would be the next option. Her resistance to RAI may be related to her predominant pattern of elevated fT3 levels. The mechanism of this is unclear but may be related to impaired RAI uptake by the thyroid gland. Future studies may be useful to evaluate this further.

PA-A-38

CHARACTERISTICS OF COVID-19 PATIENTS WITH HYPERGLYCAEMIC EMERGENCY AND MORTALITY OUTCOMES: SINGLE CENTRE EXPERIENCE IN PAHANG

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Nurbadriah Binti Jasmiad, See Chee Keong, Yung Jung Hao, Nur Liyana Binti Abdul Wahab, Nurul Zaireen Syazwana Binti Zainal
Hospital Sultan Haji Ahmad Shah Temerloh, Malaysia

INTRODUCTION

Individuals with diabetes have similar risk of contracting COVID-19 infection compared to those without diabetes. However, COVID-19 patients with diabetes are at a higher risk for severe outcomes and death. The occurrence of hyperglycaemic emergency and diabetic ketoacidosis (DKA) may worsen the outcomes of COVID-19 infection. This study will determine the characteristics of COVID-19 patients admitted with hyperglycaemic emergency and mortality outcomes in Hospital Sultan Haji Ahmad Shah, Temerloh, Pahang.

METHODOLOGY

All electronic records of COVID-19 patients admitted from March 2021 until March 2022 were reviewed for occurrence of hyperglycaemic emergency. Data regarding demographics, clinical presentation, laboratory investigations and clinical outcomes were collected. Further analysis with patients subcategorised into 2 timelines: March-December 2021 (group 1) and January-March 2022 (group 2) reflecting two surges of COVID-19 admission to the hospital was done.

RESULTS

Twenty-four COVID-19 patients with hyperglycaemic emergency [mean age 56.7 (SD 15.6) years, 54.2% female, 79.2% Malay ethnicity, 95.8% type 2 diabetes mellitus, 54.2% unvaccinated, 70.8% category 5 infection] were analysed. Majority of patients had DKA at 79.2% [mean pH 7.16(SD 0.12), mean HCO_3 10.80 (SD 3.07), mean glucose at diagnosis 25.3 (SD 11.0) mmol/L]. The mean length of hospitalisation was 11.42 (SD 7.4) days and mortality rate was 63.2%. Nine DKA cases were detected in group 1 compared to 10 cases during the shorter timeline in group 2. All patients had resolved DKA but the majority succumbed later due to complications of COVID-19 infection. Mortality rates in both groups were 66.7%(n=6) and 60%(n=6), respectively.

CONCLUSION

Despite high occurrence of uncontrolled diabetes during COVID-19 infection in this cohort, only a small proportion had hyperglycaemic emergency. In both timeline of hospitalisation surge, COVID-19 patients with concomitant hyperglycaemic emergency had poorer prognosis.

PA-A-39

A CASE OF HYPERCALCEMIA CRISIS IN PREGNANCY DUE TO GIANT PARATHYROID ADENOMA

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Eng Sy and Florence HS Tan
Department of Endocrinology, Sarawak General Hospital, Kuching, Sarawak, Malaysia

INTRODUCTION

Hypercalcemia in pregnancy affects 0.03% of reproductive women. Complications are directly related to maternal calcium level and include maternal nephrolithiasis, kidney injury, pancreatitis, pre-eclampsia and fetal loss. Primary hyperparathyroidism accounts for >90% of cases.

CASE

We report a 41-year-old female who presented at 4 weeks of gestation with a 1-week history of polyuria and 3-day history of epigastric pain.

Laboratory investigations showed the following: severe hypercalcemia with corrected serum calcium of 5.04 mmol/L, low serum phosphorous at 0.88 mmol/L, elevated intact PTH at 45.4 (NR:1.6-6.0 pmol/L), acute kidney injury with serum creatinine of 221 umol/L, and pancreatitis with serum amylase of 368 (NR: 62-106 u/L). Electrocardiogram showed Osborn waves. Kidney ultrasound showed bilateral renal medullary nephrocalcinosis with nephrolithiasis. Neck ultrasound revealed a 2.8x2.9x5.1 cm well defined solid lesion postero-inferior to the right thyroid lobe suggestive of parathyroid tumour.