CONCLUSION
Severe hypercalcaemia of pHPT can be successfully managed with aggressive treatment and close monitoring. Need for dialysis may be avoided but such patients should undergo parathyroidectomy as soon as possible.

PA-A-34

A CASE OF DENOSUMAB-INDUCE HYPOCALCEMIA:
A SEVERE AND PROLONGED CONSEQUENCES

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Mohd Hazriq A,[1,2] Sharifah Faradilla WMH,[2] Fatimah Zaherah MS, Rohana AG[2]
[1]*Hospital UiTM, Puncak Alam, Selangor, Malaysia*
[2]*Fakulti Perubatan, Universiti Teknologi MARA (UiTM), Sungai Buloh, Malaysia*

INTRODUCTION
Denosumab is known to cause abnormalities in calcium homeostasis. The majority of such cases have been described in patients with underlying metastatic cancer, chronic kidney disease or vitamin D deficiency. History of bariatric surgery could also compound the effect of hypocalcemia necessitating intravenous treatment and prolong high dose oral supplementation.

CASE
We present a 61-year-old female with a 6-day history of progressive worsening limb numbness, tingling sensation and intermittent muscle cramps. She had gastric sleeve surgery done 20 years ago. Her regular medication includes calcium, vitamin D and iron supplement. Further history uncovered a denosumab treatment for osteoporosis 1 week ago at a private hospital.

Biochemistry revealed severe hypocalcemia with adjusted calcium of 1.33 mmol/l, mild hypophosphatemia at 0.65 mmol/l, with normal magnesium and renal function. ECG showed prolonged QT interval. PTH level was high at 34.6 pmol/l and 25-OH-vitamin D was insufficient at 33 mmol/l.

She required multiple courses of intravenous calcium gluconate bolus and infusion due to retractable severe hypocalcemia while titrating up her oral supplement in the ward. She was discharged after 8 days with serum calcium around 1.90 mmol/l. At clinic follow up 5 days later, her serum calcium decreased again to 1.64 mmol/l requiring further iv calcium infusion and oral supplement adjustment.

After 2 months, she still requires high dose replacement with 1.5 ug calcitriol twice daily, 1 g calcium carbonate thrice daily and vitamin D3 replacement to maintain normocalcemia.

CONCLUSION
This case report highlights the importance of screening for risk factors for iatrogenic hypocalcemia before initiating denosumab treatment particularly for patients with a history of bariatric surgery. Vitamin D should be adequately replaced prior to treatment and serum calcium levels should be closely monitored post treatment.

PA-A-35

DOSE UP-TITRATION OF EMPAGLIFLOZIN AMONG TYPE 2 DM PATIENTS UNCONTROLLED ON EXISTING ORAL ANTIDIABETIC AGENTS

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Mohd Rahman Omar,[1] Norasyikin A Wahab,[2] Norlaila Mustafa,[2] Norlela Sukor,[2] Norazmi Kamaruddin[2]
[1]*Universiti Sains Islam Malaysia*
[2]*Universiti Kebangsaan Malaysia*

INTRODUCTION
In most trials involving empagliflozin, the effect on HbA1c reduction was based on concurrent use of 2 doses of the drug without dose titration. This study aims to determine the proportion of patients who need to up-titrate empagliflozin from 10 mg to 25 mg to achieve the desired A1c reduction.

METHODOLOGY
T2DM patients uncontrolled on existing oral glucose-lowering drugs were given empagliflozin 10 mg daily for 3 months. Those who achieved a reduction in HbA1c more than 0.5% from baseline will continue the same dose for another 3 months while those those who had HbA1c reduction of 0.5% or less will be given 25 mg daily for 3 months.

RESULTS
A total of 55 (67.9%) patients had significant HbA1c reduction >0.5% after 3 months on 10 mg empagliflozin (non-titration group), while 26 (32.1%) patients required up-titration of empagliflozin to 25 mg daily for another 3 months (up-titration group). There was no further significant reduction in mean HbA1c from 7.50% (range: 7.1 to 8.15) to 7.45% (range: 6.78 to 8.13), p=0.574 after 3 months of 25 mg empagliflozin. At 3 months therapy with empagliflozin 10 mg, 55 (67.9%) patients achieved mean HbA1c reduction of >0.5% from baseline 7.8%(range: 7.5 to 8.7) to 6.95% (range: 6.53 to 7.38), p<0.001 and remains stable after the continuation for another 3 months.

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.