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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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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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.