CONCLUSION
Managing DTC in the presence of GO represents a significant challenge to the treating physician. A multi-disciplinary approach and in-depth discussion with patients are essential in making treatment decisions.

EP_A086

MYELODYSPLASTIC SYNDROME AND GRAVES' DISEASE

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INTRODUCTION
Myelodysplastic syndrome (MDS) is a heterogeneous group of hematopoietic neoplasms characterized by bone marrow failure resulting in cytopenia and dysplastic haematopoiesis. The association between MDS and autoimmune diseases has been previously described in the literature. Here we report a case of Graves' disease (GD) with secondary MDS.

CASE
A 43-year-old male with vitiligo was admitted for pancytopenia and left parapneumonic effusion. GD was concomitantly diagnosed based on weight loss, tachycardia, exophthalmos and the presence of TSH receptor antibodies. Antithyroid drugs were carefully used with close monitoring of cell counts. Despite clinical improvement with antibiotics and achievement of biochemical control of hyperthyroidism, cytopenia persisted. Results showed WBC 1.58 to 3.38 x 10^9/L, absolute neutrophil counts 0.65 to 1.18 x 10^9/L, haemoglobin 9.6 to 10.6 g/L, and platelet counts 74 to 10^7 x 10^9/L. Autoimmune panels tested negative. Peripheral blood film revealed pancytopenia without evidence of haemolysis or blast cells. Bone marrow aspirate and trephine biopsy showed mildly hypocellular marrow with relatively reduced myelopoiesis together with subtle dysplastic changes of erythrocytes and megakaryocytes. No cytogenetic abnormality was detected.

Emerging evidence suggests that autoimmune diseases are risk factors for MDS. A Swedish population-based study demonstrated an apparent link between the development of MDS and autoimmune diseases. Most of the reported cases had a history of hypothyroidism. The development of MDS in relation to autoimmune diseases is still poorly understood. The proposed explanations include shared genetic or environmental risk factors, a direct insult to the bone marrow leading to malignant transformation by untreated autoimmune diseases, or the inflammatory process from a haematological neoplasm resulting in the subsequent diagnosis of an autoimmune disease.

CONCLUSION
Our case highlighted the association between autoimmune thyroid disease (GD) and MDS. Further studies to underpin the association and pathophysiology are required.

EP_A087

THE CALM BEFORE THE STORM AND THE STORM BEFORE THE CALM: A CASE OF RETRACTABLE THYROID STORM

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INTRODUCTION
Thyroid storm is a severe complication of hyperthyroidism with a high mortality rate. Multimodal pharmacotherapy is the cornerstone of treatment. In severe cases, plasmapheresis may also be done. However, this practice is not widespread with a lack of clear guidelines.

CASE
We describe a 37-year-old female with severe thyroid storm and multiple organ failure. She initially presented with a three-week history of worsening jaundice, dyspnea and pedal edema. At presentation, she was neurologically intact but had prominent jaundice, congestive heart failure, atrial fibrillation, goiter and mild thyroid ophthalmopathy. Tests revealed elevated FT4 >90 pmol/L, TSH <0.01 mIU/L, bilirubin 267 µmol/L and coagulopathy. With a Burch-Watforsky Point Scale of 65, full pharmacotherapy for thyroid storm was promptly instituted. Due to lack of clinical improvement and rising bilirubin, we resorted to plasmapheresis after seven days with a view for early thyroidectomy. Plasmapheresis was administered over three sessions and resulted in normalization of FT4 (14.32 pmol/L), resolution of heart failure, and improvement of bilirubin and other blood parameters. Three days later, sensorium quickly deteriorated to coma requiring intubation. EEG showed nonconvulsive seizure; other neurologic investigations were non-contributory. Her condition was further complicated with retractable arrythmia, worsening coagulopathy with lower gastrointestinal bleed, and rising FT4 level and liver function tests. Four further courses of plasmapheresis were administered to control FT4, with improvement in other biochemical markers, subsequent

resolution of seizure and recovery of consciousness. This allowed a safe window for urgent thyroidectomy four days after plasmapheresis and an uneventful surgery.

CONCLUSION

This case highlights the complexities in the management of thyroid storm, and the risk of relapse despite initial biochemical and clinical improvement. A sufficient course of plasmapheresis is essential to allow for urgent thyroidectomy.

EP_A088

ATRIAL FLUTTER IN HYPERTHYROIDISM: ACHIEVING EARLY RHYTHM CONTROL

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INTRODUCTION

Atrial arrhythmia is a common manifestation of cardiac complications of hyperthyroidism. However, most literature focus on the incidence and management of atrial fibrillation rather than atrial flutter. It also suggests postponement of cardioversion until the fourth month of maintaining a euthyroid state, as more than half of cases revert spontaneously to sinus rhythm and atrial fibrillation may recur with thyrotoxicosis. Here we present a case of atrial flutter with early rhythm control with electrical cardioversion without subsequent recurrence.

CASE

A 29-year-old male presented with a two-day history of fever and recurrent episodes of palpitations. Upon arrival, findings showed BP 110/70, temperature 38°C, and typical counterclockwise atrial flutter with variable block and HR 130 bpm on ECG. Results revealed low TSH (<0.01 mIU/L) and elevated FT4 (90 pmol/L). He was treated as Graves' thyrotoxicosis precipitated by viral fever. Treatment included carbimazole 30 mg OD, rate control with propanolol 40 mg TDS and supportive care. Echocardiogram showed EF 44% with dilated RA, RV and LA. In view of evidence of cardiomyopathy, trans-oesophageal echocardiogram and elective cardioversion was performed a week after discharge. Rhythm was successfully cardioverted back to sinus rhythm. He remains in sinus rhythm with improvement in cardiac function four weeks after cardioversion.

CONCLUSION

In general, the management of atrial flutter is slightly different from atrial fibrillation, as the former may be treated with immediate rhythm control using electrical cardioversion. Despite the thyrotoxic state, earlier rhythm control is better for cases of atrial flutter in order to prevent development or worsening of thyrotoxic cardiomyopathy.

EP_A089

PERSISTENT VOMITING AS THE PRESENTATION OF THYROTOXICOSIS: A CASE REPORT

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INTRODUCTION

Thyrotoxicosis may have many different presentations at diagnosis. We present a case of thyrotoxicosis with the presentation of only recurrent vomiting in pregnancy.

CASE

The patient's medical records were traced and reviewed.

A 41-year-old Malay female, G4P2 with one miscarriage, with underlying type 2 DM initially presented during pregnancy with recurrent admissions for vomiting since the first trimester. She was admitted and treated for hyperemesis gravidarum and urinary tract infection at 12 and 14 weeks gestation. She was readmitted at 28 weeks for recurrent vomiting and reduced oral intake one week prior to admission. She denied having conventional symptoms of thyrotoxicosis. She did not have any family history of thyroid disorder. Examination findings revealed a small goitre with no fine tremors or thyroid eye signs, BP within normal range and HR 100 to 107. Clinically, she had mild dehydration. Multiple investigations including serum calcium, ketone, amylase and brain MRI to look for the cause of persistent vomiting were normal. During her third admission, thyroid tests were done for evaluation of tachycardia. Results showed elevated FT4 (66 pmol/L), suppressed TSH (0.01mIU/L), negative thyroid antibodies and no significant abnormality on neck ultrasonography. She was started on oral carbimazole 20 mg OD and oral propranolol 20 mg BD. Vomiting was resolved thereafter. She delivered via emergency lower caesarean section at 31 weeks for abnormal cardiotocography and foetal intrauterine growth restriction.

CONCLUSION

Recurrent vomiting in pregnancy is a rare presentation for thyrotoxicosis. This should not be missed in clinical practice to prevent adverse maternal and foetal outcomes.