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.

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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