CASE

We report a case of severe cholestasis in a patient presenting with thyroid storm secondary to Graves' disease in whom heart failure and other secondary causes were appropriately investigated. We also present other relevant reports and studies available in the literature.

A 39-year-old female presented with jaundice, symptoms of thyrotoxicosis and heart failure. Clinically, she had exophthalmos with a moderately enlarged thyroid and signs of heart failure. Burch-Wartofsky Point Scale was 70. Her thyroid-stimulating hormone level was suppressed at 0.02 mU/L, with high free thyroxine of 92.4 pmol/L and free triiodothyronine of more than 30.8 pmol/L. She also had hyperbilirubinaemia which was predominantly conjugated, mildly elevated aspartate transaminase (AST) of 86 IU/L and normal alanine transaminase (ALT) level of 34 IU/L. Blood parameters were prolonged with activated partial thromboplastin time (APTT) of more than 180 seconds and international normalized ratio (INR) of 2.14. She was commenced on Lugol's iodine, corticosteroids and propranolol for thyroid storm, ursodeoxycholic acid for cholestasis and furosemide along with spironolactone for heart failure. She improved gradually and was discharged after a month of hospitalization.

CONCLUSION

Severe cholestasis in patients with thyrotoxicosis is a common presentation and may dominate the clinical picture of the primary disease. The recognition of liver and cardiac complications of thyrotoxicosis together with a thorough evaluation for other etiologies will allow proper management and hence, steady improvement of this serious medical condition.

PA-A-22

SEVERE ACUTE RESPIRATORY SYNDROME CORONAVIRUS 2 (SARS-CoV-2) VACCINE-INDUCED THYROID DYSFUNCTION: A TALE OF TWO PATIENTS

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INTRODUCTION

SARS-CoV-2 vaccine has been the main pillar in battling the coronavirus disease 2019 (COVID-19) pandemic. However, the current vast scale of SARS-CoV-2 vaccination programme has led to inevitable reports of various adverse reactions, one of which include thyroid dysfunction.

CASES

We describe two patients who manifested hyperthyroidism following BNT162b2 mRNA-based COVID-19 vaccine boosters.

Patient 1, a previously euthyroid 46-year-old female, has an eight-year history of type 1 diabetes mellitus. She developed palpitations of increasing severity about two weeks after her COVID-19 booster vaccine on 20th January 2022. She had weight loss of 4 kg and experienced menstrual irregularities in the subsequent three months. Examination revealed tachycardia (112 beats per minute, regular) and bilateral fine tremors of the hands. There was no goitre or neck tenderness. Blood investigations showed overt hyperthyroidism with positive thyroid autoantibodies, consistent with Graves' disease. Treatment with carbimazole led to marked symptomatic improvement. Patient 2, a 38-year-old female with a six-year history of Hashimoto thyroiditis, was clinically and biochemically euthyroid while taking levothyroxine 100 mcg daily prior to her COVID-19 booster vaccine on 5th January 2022. Five weeks following the vaccine, her thyroid function test during her endocrine clinic appointment showed overt hyperthyroidism, which was confirmed by a second blood sample ten days later. There was neither a change in levothyroxine dose nor any additional supplement intake. She was otherwise asymptomatic. Levothyroxine was then withheld. She regained her baseline hypothyroid state two weeks later, during which levothyroxine was resumed.

CONCLUSION

SARS-CoV-2 vaccine-induced thyroid dysfunction can affect both euthyroid and hypothyroid patients. A history of recent COVID-19 vaccination should be included in the clinical evaluation of a newly diagnosed hyperthyroid patient or unexplained hyperthyroidism in a long-standing hypothyroid patient.

PA-A-23

CHARACTERISTICS OF PATIENTS WITH TYPE 1 DM AND LADA IN A MALAYSIAN PUBLIC HOSPITAL: A CROSS- SECTIONAL STUDY

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INTRODUCTION
Type 1 diabetes mellitus (DM) occurs as the result of pancreatic beta cell destruction. Latent autoimmune diabetes in adults (LADA) is immunologically similar to T1DM but immune destruction progresses at a slower rate. The aim of the study is to identify the clinical characteristics of patients with T1DM and LADA in our clinic.

METHODOLOGY
This is a single centre cross-sectional study involving all 122 patients with T1DM and LADA. Information was obtained from patients' records and interviews during follow up.

RESULTS
There were 49 males (40.2%) and 73 females (59.8%) with a mean age of 35.3 (*SD* 14.9) years old. The mean duration of disease is 12.9 (*SD* 9.7) years. Ninety-five subjects (77.9%) have T1DM and 27 subjects (22.1%) have LADA. The most common complication was retinopathy (14.8%). Almost 2/3 of subjects (61.5%) reported having minor hypoglycemia and 15 (12.3%) had diabetic ketoacidosis in the past year. The most common co-morbid is dyslipidemia (45.9%). Eighteen percent of the subjects have other autoimmune diseases. Majority of the subjects were on at least one analogue insulin (93.4%) and on basal bolus regimen (89.3%). Only 6 subjects (4.9%) were on insulin pump. One hundred fourteen (93.4%) subjects performed self-monitoring of blood glucose (SMBG) and only 26 (21.3%) subjects have used continuous glucose monitoring systems (CGMS) at least once. The mean HbA1c is 8.91% (SD 2.2). The most frequent pancreatic autoantibodies detected were glutamic acid decarboxylase (GAD) (77.9%) and islet cell antibody (ICA) (77.7%).

CONCLUSION
Majority of our subjects with T1DM and LADA are on analogue insulin andon basal-bolus regimen with most of them performing SMBG. Despite this, the rate of hypoglycemia is high and control remains suboptimal. Increasing the use of technologies such as CGMS and insulin pumps which are not fully utilized at present, may improve outcomes.

PA-A-24

CASE REPORT: PRIMARY HYPERPARATHYROIDISM AND JAW TUMOUR SYNDROME WITH CDC73 GENE MUTATION IN A YOUNG PATIENT

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INTRODUCTION
Primary hyperparathyroidism (pHPT) occurs frequently in those over the age of 50 years. This condition is uncommon in young adults and are more likely to have an underlying germline mutation.

CASE
We present a case of a healthy 16-year-old male who was incidentally found to have an elevated calcium of 3.16 mmol/L. Family history revealed that his father, aunty and grandfather also had a history of hypercalcemia. No genetic study was done previously. The patient was diagnosed with iPTH-mediated hyperparathyroidism based on blood investigations. Localization scan revealed an overactive right parathyroid gland secreting excess iPTH. Subsequently, he was scheduled for right parathyroidectomy.

Histopathology report confirmed the diagnosis of a right superior parathyroid adenoma. His iPTH level declined from 968.5 pg/ml to 7.9 pg/ml after the surgery while calcium and ALP levels also normalized. He subsequently required calcium and activated Vitamin D supplementation.

The patient and his family were referred for further genetic assessment, revealing CDC73-related disorders, with a pathogenic mutation on CDC73 gene. The patient's father was found to develop a jaw tumour with histologic confirmation of invasive ossifying fibroma. Hence, tumour debulking was planned.

CDC73-related disorder is an autosomal dominant disorder resulting from the inactivation of the CDC73 tumor suppressor gene. The spectrum includes: Hyperparathyroidism jaw tumor (HPT-JT) syndrome, parathyroid carcinoma and familial isolated hyperparathyroidism (FIHP). Penetration of pHPT is as high as 80% to 95%, while parathyroid carcinoma may be found in more than 20% of patients. Lifelong surveillance is indicated for positive gene carriers to look for recurrent hyperparathyroidism, parathyroid carcinoma, renal and uterine tumour in females.