Evaluation in an endocrine clinic showed that he had low cortisol with low ACTH levels. Moreover, insulin tolerance test done confirms inadequate ACTH and growth hormone response. Other anterior hormonal profile results were normal. In view of the evidence of hypothalamic-pituitary-adrenal (HPA) axis suppression, coupled with the pituitary MRI findings, the diagnosis of SARS-CoV-2 vaccination-induced hypophysitis was established.

CONCLUSION

SARS-CoV-2 vaccination-induced hypophysitis is a rare but significant adverse effect that needs to be recognised. Prompt diagnosis is crucial, as treatment with steroid is lifesaving. In light of our experience, diagnosis of hypophysitis should be considered when patients present with pituitary dysfunction with a history of recent COVID-19 vaccination.

EP_A056

USTEKINUMAB-INDUCED HYPOPHYSITIS IN CROHN'S DISEASE: A CASE REPORT

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INTRODUCTION/BACKGROUND

Ustekinumab is a monoclonal antibody targeting IL-2 and IL-23 that has been used to treat psoriasis and more recently, inflammatory bowel disease. With the increased use of immunotherapy, immune-related adverse events are being reported more frequently. Ustekinumab has been reported to cause hypophysitis in a patient with psoriasis.

CASE

We report a case of a 29-year-old female with difficult-to-treat Crohn's disease since 2008. She has received azathioprine, methotrexate, infliximab, and adalimumab without favorable response. She subsequently underwent a right hemicolectomy and terminal ileal resection in 2017. She had previously been on prednisolone in 2012. She was started on ustekinumab in May 2020 and showed good clinical response. Twenty-seven months after starting ustekinumab, on routine investigation, she had a fasting blood glucose of 1.9 mmol/L. There were also some home capillary glucose readings of <3.5 mmol/L. She reported no signs of hypoglycemia and denied taking other medications or traditional supplements. She had been off all steroids for more than 10 years. She was clinically euthyroid with no history of polyuria or visual field defects. She had transient oligomenorrhea due to significant weight loss in 2020. Her BMI was 16.2. She had no signs of Cushing's. Further workup revealed low cortisol level of 43 nmol/L with ACTH of 11 pg/ml. TFT was discordant with elevated FT4 of 34 pmol/L and normal TSH of 2.69 mIU/L, with no assay interference confirmed. Her prolactin level was normal at 374 mIU/L. Pituitary MRI showed loss of posterior pituitary bright spot, slightly thickened stalk with heterogeneity within the pituitary suggesting possible hypophysitis. She started hydrocortisone replacement and her hypoglycemia resolved. As she is currently responding well to the ustekinumab for her Crohn's, she will be monitored periodically for progression of her hypophysitis.

CONCLUSION

Patients treated with ustekinumab should be monitored periodically for autoimmune endocrinopathies such as hypophysitis and thyroiditis.

EP_A057

A RARE CASE OF MICROPROLACTINOMA AND GRANULOMATOUS MASTITIS

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INTRODUCTION/BACKGROUND

Idiopathic granulomatous mastitis (IGM) is a benign and rare chronic inflammatory disease of the breast. However, its clinical presentation can mimic a breast malignancy or abscess. The aetiology is often unknown but several predisposing factors were identified, including patients with autoimmune disorders, hyperprolactinemia secondary to pregnancy, lactation, dopamine antagonist usage or pituitary adenoma.

CASE

We report a rare case of IGM in a patient with hyperprolactinemia secondary to microprolactinoma.

A 45-year-old, para 2, premenopausal female presented to a surgical clinic with a 2-year history of intermittent bilateral galactorrhoea and right breast swelling with pus discharge. There was no menstrual irregularity. Ultrasound of the right breast demonstrated multiloculated hypoechoic collections with internal echogenicities. She was treated with antibiotics; however, there was no clinical

improvement, and she underwent biopsy of the lesion which revealed chronic granulomatous inflammation, confirming the diagnosis of IGM. She was also found to have hyperprolactinemia at 125.9 ng/ml secondary to a microprolactinoma and was started on cabergoline. Two weeks post treatment, the prolactin level normalized with resolution of galactorrhoea.

CONCLUSION

Hyperprolactinemia is one of the predisposing factors for the development of IGM by increasing inflammation of the breast tissue. When evaluating for IGM, serum prolactin should always be measured to exclude elevated prolactin levels. The cause of hyperprolactinemia should be further investigated and addressed, and treatment with dopamine receptor agonist could reduce recurrence of IGM.

EP_A058

FASTING AND POSTPRANDIAL HYPOGLYCEMIA IN AN ADOLESCENT PRESENTING WITH ENDOGENOUS HYPERINSULINEMIC HYPOGLYCEMIA LIKELY INSULINOMA: A CASE REPORT

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INTRODUCTION/BACKGROUND

Hypoglycaemia is an uncommon clinical problem in individuals without diabetes mellitus. It is a clinical syndrome with various causes in which low plasma glucose leads to hypoglycaemic symptoms and signs that resolve when the plasma glucose is raised. The diagnosis of a true hypoglycaemic disorder requires fulfilment of these specific criteria (Whipple's triad). Once the presence of a hypoglycaemic disorder is verified, a detailed clinical history often suggests a specific underlying cause. Insulinoma is a type of functional neuroendocrine tumour characterized by hypersecretion of insulin, causing hypoglycaemia, characteristically fasting hypoglycaemia. We describe a patient with insulinoma with both fasting and postprandial hypoglycaemia.

CASE

A pre-morbidly healthy 12-year-old male presented with recurrent hypoglycaemia for 3 months. He experienced a severe episode of hypoglycaemia manifesting as seizure with blood glucose of 1.4 mmol/L. Symptoms resolved after glucose administration. He developed progressive weight gain over the course of 4 years. His BP was 120/72, PR 98, BMI 30 with absence of acanthosis nigricans. Blood investigation during clinic visit revealed asymptomatic hypoglycaemia mediated by endogenous hyperinsulinemia, with random blood glucose of 1.7 mmol/L (<3 mmol/L), serum insulin 364.8 pmol/L (>20 pmol/L) and C-peptide 1495 pmol/L (>200 pmol/L). Mixed meal test confirmed fasting hypoglycaemia, with RBS of 2.5 mmol/L, insulin 300 pmol/L, C-peptide of 1145pmol/L. At 120 minutes (postprandial) following the test, RBS was 2.3 mmol/L, insulin 557 pmol/L, C-Peptide 2282 pmol/L. Prolonged supervised fasting test revealed hypoglycaemia after 4 hours with RBS 2 mmol/L, insulin 286.8 pmol/L and C-peptide 1459 pmol/L. Beta-Hydroxybutrate remained suppressed at 0.1 mmol/L following fasting. Sulphonylureas screening was negative. HbA1c was 4.2% and serum Ca (corrected) was 2.22 mmol. CT pancreatic protocol revealed a hypervascular lesion (1.9 x 2.0 x 2.9cm) at the pancreatic head. Diazoxide was initiated to prevent hypoglycaemia and it was well tolerated. The patient is planned for surgical resection of the pancreatic lesion which is likely to be an insulinoma.

CONCLUSION

Recurrent hypoglycaemia requires careful and comprehensive assessment to diagnose a patient. Around 20% of insulinoma patients have both fasting and postprandial hypoglycaemia needing clinical suspicion and prompt assessment to improve outcomes for these patients.

EP_A059

LONG-STANDING ACROMEGALY WITH PERSISTENT DISEASE RESPONSIVE TO PASIREOTIDE: A CASE REPORT

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INTRODUCTION/BACKGROUND

In acromegaly patients, chronic hypersecretion of growth hormone from pituitary adenoma results in significant morbidity and mortality. Achieving biochemical control can be challenging, requiring a combination of pituitary surgery, radiotherapy and medical therapy. Pasireotide, a new multireceptor-targeted somatostatin receptor ligand, has a broader binding profile and an increased affinity for SSTR1, 2, 3, and 5 that has demonstrated superiority compared to Octreotide LAR.