prolactin (35.7 mIU/L) and low IGF-1 level of 11.9 ng/ml (69 -227 ng/ml) suggestive of growth hormone deficiency. Additionally, her vitamin D level was insufficient, 68.79 nmol/L. Short Synacthen test revealed adequate cortisol response. MRI of pituitary reported features of empty sella, confirming the diagnosis of primary ESS. She received hormonal replacement therapy, including estradiol, for pubertal induction. She was counselled for right total hip replacement, but she was not keen.

CONCLUSION

Panhypopituitarism observed in ESS affects bone remodeling, leading to early osteoporotic fracture. Treatment with hormonal replacement is essential to restore secondary sexual characteristics for psychosocial well-being as well as to improve bone health to reduce the risk of further fracture.

EP_A043

RACING HEART UNDER STORMY SKIES: A JOURNEY THROUGH AGRANULOCYTOSIS TO THYROID STORM

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INTRODUCTION/BACKGROUND

Thyroid storm is a rare but life-threatening exacerbation of thyrotoxicosis characterised by multi-system dysfunction. Its shared features with sepsis may pose a significant diagnostic challenge. We describe a patient with Graves' disease who developed carbimazole-induced agranulocytosis followed by thyroid storm necessitating therapeutic plasma exchange (TPE) and urgent thyroidectomy.

CASE

A 31-year-old female with newly diagnosed Graves' disease on carbimazole 40 mg daily presented with palpitations and right hypochondriac pain. Initial investigation showed elevated FT4 at 31 pmol/L (7.86-14.41 pmol/L), suppressed TSH at 0.03 uIU/mL (0.38-5.33 uIU/mL) with cholestatic transaminitis. On day 3 of admission, she developed agranulocytosis with an absolute neutrophil count (ANC) of 0.06 x 10^9/L, hence carbimazole was withheld. Granulocyte-colony stimulating factor was initiated along with a broad-spectrum antibiotic to cover for neutropenic sepsis. The ANC normalised after three days, but she developed spiking fever up to 40.8°C, associated with persistent vomiting followed by hypotension and tachycardia. Biochemical tests revealed rising FT4 to 44.5 pmol/L and hyperbilirubinemia. Initiation of glucocorticoids upon withholding carbimazole was delayed due to concern of sepsis. Diagnosis of thyroid storm was made and urgent TPE was initiated along with high-dose intravenous glucocorticoids and esmolol infusion. She showed immediate clinical improvement with defervescence and stabilization of hemodynamic parameters after the first cycle of TPE. Total thyroidectomy was performed after two cycles of TPE, and she was discharged well on day 30.

CONCLUSION

This case highlights the challenge of distinguishing thyroid storm from sepsis in the setting of carbimazole-induced agranulocytosis. It underscores the importance of prompt recognition and timely intervention of thyroid storms to prevent morbidity and mortality. Early initiation of TPE as bridging therapy before definitive therapy in the setting where antithyroid drug was contraindicated provided rapid control of thyrotoxicosis and was well tolerated.

EP_A044

THROUGH THE EYES OF LUPUS: LIPAEMIA RETINALIS AS A RARE OCULAR MANIFESTATION OF HYPERTRIGLYCERIDEMIA

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INTRODUCTION/BACKGROUND

Lipaemia retinalis is a rare but striking ocular finding caused by extreme hypertriglyceridemia. It is typically associated with primary dyslipidemias but may also occur secondary to autoimmune disease such as systemic lupus erythematosus (SLE). We describe a case of newly diagnosed SLE with lupus nephritis, incidentally found to have lipaemia retinalis, leading to the diagnosis of severe hypertriglyceridemia.

METHODOLOGY

A 12-year-old female presented with two months of intermittent fever and constitutional symptoms. Investigations revealed normochromic normocytic anemia, raised inflammatory markers, positive ANA (speckled pattern) and anti-dsDNA with low complement levels. Proteinuria was present, and subsequent renal biopsy confirmed class III lupus nephritis. Fundoscopy revealed an incidental finding of lipaemia retinalis, and lipid profile showed severe

Adult E-Poster

hypertriglyceridemia with a serum triglyceride (TG) of 11.7 mmol/L. Other secondary causes of hypertriglyceridemia, including hyperglycemia, hypothyroidism, alcohol or dietary fat, were excluded from biochemistry and clinical history. The lipid profiles of first-degree relatives were unremarkable.

She was started on high-dose omega-3 fish oil, a very low-carbohydrate diet and fenofibrate 145 mg every other day along with immunosuppression therapy for SLE. Her serum TG dropped markedly to 4.1 mmol/L within 3 weeks. However, she had transient bradycardia leading to temporary cessation of fenofibrate and hydroxychloroquine, and her serum TG rebounded to 16.5 mmol/L. After ruling out other causes of bradycardia, fenofibrate was resumed without recurrence of bradycardia, followed by normalization of the TG level. Fundoscopic examination two months later showed resolution of lipaemic retinalis. She completed six cycles of cyclophosphamide with steroid tapering, and her serum TG remained normal at 0.5 mmol/L.

CONCLUSION

This case highlights lipaemia retinalis secondary to severe hypertriglyceridemia as a rare manifestation in newly diagnosed SLE. Early recognition, aggressive lipid-lowering therapy, along with immunosuppressive treatment for the underlying SLE led to rapid triglyceride reduction with complete resolution of lipemia retinalis.

EP_A045

WEIGHT REBOUND POST GLP-1 RA CESSATION: THE IMPORTANCE OF GRADUAL TAPERING AND PATIENT EDUCATION

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INTRODUCTION/BACKGROUND

Glucagon-like peptide-1 receptor agonists (GLP-1 RAs) have demonstrated significant efficacy in weight management. However, abrupt discontinuation often leads to an uncontrollable appetite rebound and subsequent weight regain. This phenomenon underscores the need for structured tapering protocols and comprehensive patient education to ensure sustainable weight management post-therapy cessation.

CASE

A 54-year-old female with obesity (BMI 33.8) was initiated on subcutaneous Saxenda (liraglutide) for weight management. She successfully escalated to a 3 mg daily dose, tolerating mild gastrointestinal side effects. Despite an initial weight reduction (94.5 kg >92.4 kg), she discontinued treatment due to injection site reactions. Three months post-discontinuation, her weight increased to 96.7 kg (BMI 35.95) with increased appetite and dietary non-compliance. Upon restarting therapy, a gradual dose escalation was advised to minimize adverse effects and improve adherence. The patient also received structured education on medication tapering, dietary modifications, and lifestyle interventions.

CONCLUSION

This case highlights the challenges of abrupt GLP-1 RA discontinuation and the subsequent weight rebound. A strategic tapering plan is essential to mitigate appetite dysregulation and sustain weight loss. Moreover, patient education on the physiological impact of cessation, proper injection techniques, and behavioral strategies is crucial in optimizing long-term obesity management outcomes. Health practitioners must emphasize these aspects to ensure adherence and enhance treatment success.

EP_A046

FROM PANIC DISORDER TO CARCINOID SYNDROME IN AN EXPECTING MOTHER

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INTRODUCTION/BACKGROUND

Carcinoid syndrome occurs in ~10% of neuroendocrine tumours (NET). It indicates advanced disease with liver metastasis associated with lower survival. However, it is often misdiagnosed as other gastrointestinal, respiratory or dermatologic conditions, with a median delay in diagnosis of 3.4 years because of its rarity.

CASE

We present a case of a 32-year-old female at 10 weeks gestation presenting with abdominal distension. Physical examination revealed hepatomegaly and a pansystolic murmur. Ultrasound showed an enlarged liver with multiple solid lesions. Liver biopsy confirmed a well-differentiated grade 2 NET. Further history revealed a 2-year history of progressive facial flushing and diarrhoea that had been diagnosed as panic attacks. Endoscopic ultrasound showed