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.

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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

a 2.3 cm pancreatic mass. Echocardiography showed moderate tricuspid regurgitation with an enlarged right atrium. A provisional diagnosis of carcinoid syndrome secondary to pancreatic NET with liver metastases was made. A multidisciplinary team decision was made to terminate the pregnancy to allow further evaluation and treatment. Computed tomography of the thorax, abdomen and pelvis confirmed the pancreatic head lesion with liver metastasis. Biochemistry showed elevated 24-hour urinary 5-hydroxyindoleacetic acid and serum chromogranin A, confirming the diagnosis of carcinoid syndrome. Histopathology of the biopsied pancreatic mass was consistent with grade 2 NET with a Ki-67 of 3-4%. Gallium-68 DOTATE and fluorodeoxyglucose positron emission tomography demonstrated concordant disease involving the pancreatic head, liver, lymph nodes and bone. The tumour was deemed inoperable and the patient was commenced on somatostatin analogue, followed by peptide receptor radionuclide therapy given the predominant Gallium-68 DOTATE-avid disease.

CONCLUSION

This case highlights the delay in diagnosis of carcinoid syndrome due to the lack of awareness of NET, leading to a heavy, inoperable tumor burden with guarded prognosis. A concerted effort is required to educate all healthcare providers on NET to minimise delay in diagnosis and improve patient outcomes.

EP_A047

HABBATUS SAUDA OIL-INDUCED SEVERE HYPERTRIGLYCERIDEMIA IN A PATIENT WITH DIABETES MELLITUS

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INTRODUCTION/BACKGROUND

Hypertriglyceridemia (HTG) is a significant risk factor for cardiovascular disease, pancreatitis, and metabolic complications, particularly in patients with multiple comorbidities. While herbal supplements like Habbatus Sauda (*Nigella sativa*) oil are often used for their purported health benefits, emerging reports suggest potential adverse metabolic effects. This case highlights severe HTG potentially triggered by Habbatus Sauda oil in a patient with T2DM, dyslipidaemia and psoriatic arthritis (PsoA).

CASE

A 50-year-old Malay male, a research officer, presented with poorly controlled diabetes (HbA1c: 11%), dyslipidemia and severe HTG. He had a five- to six-year history of T2DM, previously on oral hypoglycemic agents (OHA) but discontinued, along with hypertension and PsoA managed with methotrexate (MTX). His triglyceride (TG) levels fluctuated significantly (2.1 → 13 → 8 mmol/L), with worsening levels temporally associated with the consumption of Habbatus Sauda oil. No other dietary or medication changes could fully explain the lipid surge.

The patient was advised to discontinue Habbatus Sauda oil and implement strict lifestyle modifications. Pharmacological interventions included metformin XR, gliclazide MR, vildagliptin, dapagliflozin (self-purchased), fenofibrate and atorvastatin. Despite adherence challenges, TG levels improved from 13 mmol/L to 8 mmol/L following supplement cessation and medication optimization. Hyperkalemia (K: 6.0 mmol/L) was incidentally detected, requiring urgent potassium-lowering therapy. The patient remained resistant to injectable anti-diabetic therapy and exhibited inconsistent compliance with diet and medications.

CONCLUSION

This case highlights a potential link between Habbatus Sauda oil and worsening hypertriglyceridemia, underscoring the need for vigilance in patients with pre-existing metabolic disorders. While herbal supplements are widely perceived as beneficial, they may have unintended metabolic consequences, particularly in high-risk individuals. Clinicians should actively inquire about supplement use when evaluating unexplained dyslipidemia and provide comprehensive patient education on adherence and supplement safety. A multidisciplinary approach is essential to optimizing long-term cardiovascular and metabolic outcomes.