Adult E-Poster

EP_A030

HYPERTHYROIDISM WITH SEVERE TRANSAMINITIS IN A PREGNANT FEMALE WITH A TOXIC NODULE

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INTRODUCTION/BACKGROUND.

Hyperthyroidism occurs in 0.1–0.4% of pregnancies, mostly due to Graves' disease. Other causes include toxic adenoma, multinodular goitre and trophoblastic tumours.

CASE

A 27-year-old primigravida at 9 weeks presented with vomiting, epigastric discomfort, anorexia, palpitations and weight loss of 8 kg in one month. Family history was significant for thyroid malignancy. On examination, she was alert but dehydrated, icteric and tachycardic (HR 120 bpm), spiking fever (38°C) with tremors present. No goitre or thyroid eye signs noted. Systemic findings were unremarkable. Investigations revealed TWC: 13 x10⁹/L, Urea: 8 mmol/L, Sodium: 119 mmol/L, TB: 31.5 µmol/L, ALP: 74 U/L, ALT: 943 U/L and AST: 630 U/L. ECG showed sinus tachycardia. She was managed symptomatically with anti-emetics and intravenous fluids. However, her liver function worsened; TB: 56 µmol/L, ALP: 69 U/L, ALT: 1583 U/L and AST: 530 U/L. TFT revealed thyrotoxicosis with Free T4 >100 pmol/L and TSH 0.012 mIU/L. Neck ultrasonography showed a homogenous, non-enlarged thyroid gland with a TIRADS 5 thyroid nodule on the right. Normal vascularity was seen within the gland. Hepatic ultrasonography was unremarkable. She was initiated on oral propranolol 60 mg TDS and Lugol's iodine 10 drops TDS for five days. Thyroid autoantibodies, viral hepatitis panel and other second-line investigations for transaminitis were negative. As results improved, she was discharged with tapered Lugol's iodine (5 drops TDS for 5 days) and continued on propranolol. After two weeks, both liver and thyroid function normalised. Fine-needle aspiration cytology of the thyroid nodule revealed atypia of undetermined significance, Bethesda Category III. Postpartum hemi- or total thyroidectomy has been planned as she remains well with a stable pregnancy.

CONCLUSION

Significant transaminitis in early pregnancy mandates a broad differential, including thyroid dysfunction, as early recognition ensures favourable maternal and fetal outcomes. In selected cases, short-term iodine and beta-blockade offer a safe, effective bridge to definitive management.

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FLORID ERUPTIVE XANTHOMAS IN A FAMILIAL HYPERTRIGLYCERIDEMIA PATIENT

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INTRODUCTION/BACKGROUND

Eruptive xanthomas can be an early indicator of lipid disorders, occurring in approximately 8.5% of patients with severe hypertriglyceridemia.

CASE

A 27-year-old Malay male, with a history of Type 2 Diabetes Mellitus and Dyslipidemia since age 19, presented for treatment after being lost to follow-up. He reported osmotic symptoms, weight loss and recurrent severe epigastric pain radiating to the back. He is a teetotaler, heavy smoker (13 pack-years), with poor dietary habits and frequent high-fat, high-carbohydrate meals. Family history includes diabetes and dyslipidemia in both parents.

On examination, BMI was 27 kg/m², waist circumference 80 cm and blood pressure 144/91 mm Hg. Crops of 2–5 mm yellow nodules were present over the elbows, buttocks, knees, lateral malleolus, and interphalangeal joints, along with yellowish papules over palms and back, suggestive of eruptive xanthomas. There was no arcus senilis or xanthelasma. Other systemic examinations were unremarkable. Fundoscopy showed no lipemia retinalis or diabetic retinopathy.

Investigations revealed markedly elevated Total Cholesterol (TC) 15.89 mmol/L, Triglycerides (68.44 mmol/L), low HDL (0.61 mmol/L), with invalid LDL values. HbA1c was 10.7%, with mild transaminitis, proteinuria and CKD stage 2 (eGFR 61 mL/min/1.73 m²). Amylase, TSH and other biochemical tests were normal. ECG and echocardiogram were normal. Abdominal imaging revealed fatty liver without chronic pancreatitis.

He was admitted for triglyceride lowering with IV insulin infusion and started on a low-calorie diabetic diet. Medications included Dapagliflozin 10 mg OD, Metformin 1 g BD, Rosuvastatin 40 mg ON, fenofibrate 145 mg ON and Omega-3 fatty acids 3600 mg/day. At one-month follow-up, lipid levels improved (TC 3.81 mmol/L, TG 8.95 mmol/L, HDL 0.8 mmol/L), though LDL remained invalid. He is planned for PCSK9 inhibitor initiation and was referred to dermatology for xanthomas. Genetic testing is also scheduled.

CONCLUSION

Early recognition and management of severe hypertriglyceridemia is vital to reduce risks of acute pancreatitis as well as long-term cardiovascular complications.

EP_A032

MULTIMODAL MANAGEMENT OF METASTATIC INSULINOMA: A CASE REPORT

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INTRODUCTION/BACKGROUND

Pancreatic neuroendocrine tumors (pNETs) are rare, with insulinomas being the most common functional variant. Malignant insulinomas, constituting only about 10% of cases, pose significant management challenges due to refractory hypoglycemia and limited treatment options.

CASE

We present a case of a 58-year-old male with metastatic insulinoma who required a multifaceted approach to control severe, recurrent hypoglycemia. Initial investigations revealed a pancreatic head tumor with liver metastases. Despite medical therapy with diazoxide, octreotide and verapamil, the patient remained dependent on dextrose infusions. Multidisciplinary input guided the initiation of sequential local and systemic therapies, including radiofrequency ablation (RFA), transarterial chemoembolization (TACE), and peptide receptor radionuclide therapy (PRRT). These interventions improved glycemic stability, allowing for eventual weaning off dextrose infusions. He was subsequently initiated on capecitabine and temozolomide for systemic disease control.

Malignant insulinomas necessitate an individualized, multimodal approach. In this case, aggressive local tumor control strategies in combination with systemic therapies successfully mitigated hypoglycemic episodes and improved the patient's quality of life. This report highlights the importance of early multidisciplinary intervention in optimizing outcomes for metastatic insulinoma patients.

CONCLUSION

Metastatic insulinoma remains a rare but highly morbid entity. A comprehensive, multimodal strategy integrating medical, interventional, and systemic therapies is essential to manage refractory hypoglycemia and tumor progression. This case underscores the need for early referral to specialized centers for optimal patient outcomes.

EP_A033

INITIATION OF CARBIMAZOLE WHEN BASELINE LIVER TRANSAMINASES ARE 3 TO 5 TIMES OF UPPER LIMIT OF NORMAL: A DIRE CLINICAL JUDGEMENT OR AN EVIDENCE-BASED PRACTICE

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INTRODUCTION

Antithyroid drugs (ATDs) are the first-line treatment options for hyperthyroidism. ATDs are generally avoided when transaminases are >3-5 times the upper limit of normal. We present a case of carbimazole initiation despite transaminitis of almost 5 times the upper limit of normal (ULN).

CASE

A 69-year-old female with underlying hypertension presented with a 2-week history of worsening palpitations, poor oral intake, lethargy and vomiting. Upon review, her vital signs were stable except for a heart rate of 160 beats/min. Physical examination demonstrated warm peripheries and fine tremors. ECG revealed atrial fibrillation. IV Propranolol 1 mg was given, and the rhythm reverted to sinus. Initial blood tests showed overt hyperthyroidism, FT4 >78 pmol/L and suppressed TSH <0.005 uIU/ml. Her baseline transaminases were elevated at ALT 231 U/L (5-49 U/L), AST 162 U/L (4-39 U/L), with normal serum ALP and total bilirubin. Since liver transaminases were raised, ATD was not started, but Lugol's iodine 10 drops thrice daily and Propranolol 40 mg TDS were given. Hepatobiliary ultrasound showed fatty liver disease, while neck ultrasound showed features of Graves' disease. Static ALT readings of 203 U/L and 237 U/L were recorded later. Lugol's iodine was discontinued, and T carbimazole 10 mg