

Adult E-Poster

BD was started. Close monitoring of transaminases was done. One week later, during clinic review, she was well and her ALT improved to 136 U/L with AST 71 U/L, ALP 63 U/L, and total bilirubin level 12 umol/L.

CONCLUSION

According to the American Thyroid Association (ATA), patients with transaminases >5 times the ULN should reconsider before initiating ATDs. However, ATDs can be cautiously trialed in such patients with transaminitis, provided liver function is closely monitored. In such circumstances, methimazole is recommended over PTU due to reduced hepatotoxicity risk.

EP_A034

A MULTIPRONGED APPROACH TO ACHIEVE SIGNIFICANT LDL CHOLESTEROL REDUCTION: A CASE FROM A METABOLIC CLINIC

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INTRODUCTION/BACKGROUND

Lowering low-density lipoprotein cholesterol (LDL-C) is crucial in reducing cardiovascular disease (CVD) risk, especially in patients with metabolic syndrome and obesity. While statins remain the primary pharmacological intervention, a comprehensive approach incorporating lifestyle changes and adjunctive therapies can yield remarkable results. This case highlights the successful application of a multipronged strategy in a metabolic obesity clinic.

CASE

A 38-year-old Malay female with obesity, type 2 diabetes mellitus (T2DM), dyslipidemia, and fatty liver was followed up for lipid management. Upon her initial visit to the metabolic obesity clinic two years ago, her LDL-C was markedly elevated at 5.7 mmol/L. She was started on atorvastatin 20 mg nightly alongside lifestyle modifications.

To further improve metabolic control, Contrave (naltrexone-bupropion) was introduced initially for weight management but was sequentially switched to Rybelsus (oral semaglutide) over the past year. A structured dietary approach, including a low-calorie diet with reduced refined carbohydrates and increased fiber intake, was implemented along with gradual exercise initiation.

Over two years, her LDL-C dropped dramatically from 5.7 mmol/L to 1.6 mmol/L. Concurrently, triglycerides improved, HDL-C increased, and her HbA1c decreased from 7.2% to 5.6%. She also achieved clinically significant weight loss, from 91 kg to 86 kg. This comprehensive intervention led to substantial cardiometabolic benefits.

CONCLUSION

This case demonstrates that a multipronged approach integrating statins, novel glucose-lowering agents and lifestyle modifications can achieve exceptional LDL-C reduction and broader metabolic improvements. Clinicians should consider a patient-centered, holistic strategy to optimize lipid control and long-term cardiovascular outcomes.

EP_A035

FAHR'S SYNDROME SECONDARY TO NON-SYNDROMIC PRIMARY HYPOPARATHYROIDISM

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INTRODUCTION/BACKGROUND

Fahr's syndrome is a rare neurological disorder characterized by abnormal calcium deposits in the brain, particularly in the basal ganglia. The aetiology can be primary or secondary, with endocrinopathies being the most common cause. We report a case of Fahr's syndrome in which the patient developed seizures and ECG changes due to severe hypocalcemia.

CASE

A 29-year-old female with underlying type 2 diabetes, psoriasis, and cognitive delays presented with an episode of generalized tonic-clonic seizure along with perioral numbness, skin redness and peeling for one week. Medical records showed her corrected calcium was less than 1.9 mmol/L for over a decade. There was no history of neck surgery or radiation, nor similar conditions in her family. She had no dysmorphic features but was septic with a capillary glucose of 29.5 mmol/L. ECG revealed prolonged QT interval of 516 Msec. Laboratory results showed profound hypocalcemia of 1.28 mmol/L, hypomagnesemia

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of 0.65 mmol/L and normal phosphate level. Intact PTH and 25-hydroxy-vitamin D levels were low, at 0.485 pmol/L and 24.5 nmol/L, respectively. Her brain CT scan showed cerebral atrophy with extensive intracranial calcifications, features which were consistent with Fahr's syndrome. Other evaluations did not suggest infiltrative or autoimmune disorders. There was no cataract or nephrolithiasis as a result of prolonged hypocalcemia. A multidisciplinary team managed her in the ICU with a diagnosis of severe sepsis secondary to erythrodermic psoriasis with superimposed bacterial infection. One week later, she was discharged well with calcium carbonate 1 gram thrice daily and calcitriol 0.5 mcg twice daily. No genetic test was performed due to financial constraints.

CONCLUSION

This case underscores the importance of timely diagnosis of primary hypoparathyroidism to prevent long-term complications. There are no established guidelines for the radiological surveillance intervals in Fahr's syndrome, and individualized management remains crucial in caring for patients with this condition.

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PEMBROLIZUMAB INDUCED DIABETES MELITUS IN AN ELDERLY WOMEN WITH NON-SMALL CELL LUNG CANCER

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INTRODUCTION/BACKGROUND

Immune checkpoint inhibitor (ICI)-induced diabetes mellitus is rare, with an incidence of 0.9 to 2%. As ICI usage increases, awareness of associated endocrinopathies, particularly diabetes, is crucial.

CASE

We describe a rare case of a 72-year-old non-diabetic female with NSCLC (non-small cell lung carcinoma) who presented with diabetic ketoacidosis after initiation of an immune checkpoint inhibitor.

Diagnosed with advanced NSCLC in 2023, she enrolled in a clinical trial and received a three-weekly regimen which included Pembrolizumab. She completed three cycles without major side effects, with fasting blood glucose between 5–6 mmol/L.

During her fourth trial visit, she complained of lethargy, with a glucometer reading of 28 mmol/L. Further testing

indicated diabetic ketoacidosis. She was hospitalized and started on the standard DKA fluid and insulin regimen. She was phenotypically lean, with no evidence of insulin resistance, and HbA1c taken at the time was 6.9%, indicating the glucose spike to be recent. Controlling her glucose levels in the ward was challenging. Eventually, despite resolution of DKA, she required high insulin doses (>1 u/kg/day) upon discharge.

Blood investigation at the time did not show evidence of other endocrinopathies, renal or liver impairment, and pancreatic enzymes were not significantly elevated. Her insulin autoantibody tests (ICA/anti-GAD/IAA) were negative. However, her C-peptide levels were markedly depleted at <6.67 pmol/L, indicating absolute endogenous insulin deficiency. After three cycles of ICI, repeated scans showed progression of her disease, and she was eventually withdrawn from the clinical trial. Her diabetes persisted despite cessation of her immunotherapy, requiring lifelong insulin.

CONCLUSION

The onset of ICI-induced diabetes here aligns with the reported median presentation times. Anti-PD-1 immune events are not contraindications and correlate with better progression-free survival. However, insulin therapy is often lifelong, highlighting the importance of early detection, prompt insulin initiation and regular endocrinopathy monitoring in affected patients.

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PEMBROLIZUMAB-INDUCED HYPOPHYSITIS WITH CENTRAL DIABETES INSIPIDUS: A RARE IMMUNE-MEDIATED ADVERSE EVENT

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INTRODUCTION/BACKGROUND

With the growing use of immune checkpoint inhibitors, hypophysitis is gaining increased clinical recognition while remaining a formidable diagnostic and therapeutic challenge. Pembrolizumab, a PD-1 inhibitor, is a breakthrough therapy that enhances the immune system's attack on tumours but comes with the risk of immune-related adverse events.