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.

EP_A036

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.

EP_A037

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 break-through therapy that enhances the immune system's attack on tumours but comes with the risk of immune-related adverse events.