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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Dr Norisha Nandini
Hospital Pulau Pinang, Malaysia

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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Mohd Fyzal Bahrudin,[1,2] Tharsini Sarvanandan,[1] Nicholas Ken Yoong Hee[1]
[1]Department of Medicine, University Malaya Medical Centre, Malaysia
[2]Department of Medicine, Universiti Putra Malaysia, Serdang, Selangor, Malaysia

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.

Adult E-Poster

CASE

We report the case of a 65-year-old male undergoing pembrolizumab treatment for renal cell carcinoma who presented with profound lethargy 18 months after treatment initiation. Hormonal evaluation upon admission revealed panhypopituitarism, characterized by critically low random cortisol (<14 nmol/L), ACTH deficiency (<5 pg/mL), and biochemical findings suggestive of secondary hypothyroidism (TSH: 1.68 mIU/L [0.55–4.78], free T4: 10.7 pmol/L [11.5–22.7]). The gonadal function was preserved (testosterone: 21.5 nmol/L; LH: 5.5 IU/L [1.5–9.3]; FSH: 15.8 IU/L [1.4–18.1]), while prolactin levels were mildly elevated (315 mIU/L). The autoimmune screening was ANF positive but only with titre 1:80, normal anti-dsDNA, and normal C3C4 and tumour markers were unremarkable.

The patient was promptly initiated on intravenous hydrocortisone, followed by a tapering regimen of oral hydrocortisone and thyroxine replacement. Shortly after glucocorticoid initiation, he developed polyuria and polydipsia. Further evaluation confirmed cranial diabetes insipidus (DI), with low urine osmolality (101 mOsm/kg) and elevated serum osmolality (287 mOsm/kg). Subcutaneous desmopressin was initiated, leading to rapid symptom resolution and stabilization. A pituitary MRI showed no evidence of adenoma or stalk enlargement. Although pembrolizumab-induced hypophysitis is a known immune-related adverse event, arginine vasopressin (AVP) deficiency remains a rare complication of checkpoint inhibitor therapy.

CONCLUSION

This case highlights the spectrum of pembrolizumab-induced hypophysitis, which can manifest as panhypopituitarism and, in rare cases, cranial diabetes insipidus. Clinicians should maintain a high index of suspicion for hypophysitis in patients with new-onset fatigue post-ICI therapy, as timely hormonal replacement is crucial in preventing life-threatening adrenal insufficiency and associated complications.

EP_A038

DIABETIC MASTOPATHY IN A PATIENT WITH TYPE 1 DIABETES MELLITUS

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Fei Bing Yong,[1] Chun How Phan,[1] Phei Fern Wang,[2] Jean Mun Cheah,[1] Xin Yi Ooi,[1] Hui Chin Wong,[1] Sy Liang Yong[1]

[1]*Department of Internal Medicine, Hospital Tengku Ampuan Rahimah, Klang, Malaysia*

[2]*Department of Pathology, Hospital Tengku Ampuan Rahimah, Klang, Malaysia*

INTRODUCTION/BACKGROUND

Diabetic mastopathy is a rare fibroinflammatory condition that predominantly affects long-standing type 1 diabetes mellitus. It commonly presents as firm and painless breast masses, mimicking malignancy. The diagnosis is often based on clinical evaluation, imaging studies and pathological correlation. While the exact pathophysiology remains unclear, it is hypothesized to involve an autoimmune mechanism, leading to lymphocytic infiltration and stromal fibrosis in the breast tissue.

CASE

We present the case of a 30-year-old primigravid at 16 weeks of gestation, with a background of poorly controlled long-standing type 1 diabetes mellitus complicated by diabetic nephropathy and retinopathy. She presented with a painless lump in her left breast. Clinical examination found a 3 × 2 cm mass in the upper outer quadrant of the left breast, which was firm, mobile and non-tender. There were no overlying skin changes. Breast ultrasound revealed multiple irregular hypoechoic masses with pronounced posterior shadowing. Histopathological examination (HPE) of the mass showed dense stromal keloidal type fibrosis with moderate lymphocytic infiltration around the periductal, peri-lobular and perivascular regions. The diagnosis of diabetic mastopathy was made, and reassurance was given to the patient.

CONCLUSION

Diabetic mastopathy is a benign self-limiting breast lesion with a high risk of recurrence after surgical intervention, hence it generally does not require treatment. The awareness of this rare condition may avoid unnecessary surgical intervention, mental distress, as well as diagnostic uncertainty.