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.

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DIABETIC MASTOPATHY IN A PATIENT WITH TYPE 1 DIABETES MELLITUS

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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.