Choosing the one-step IADPSG criteria's for GDM screening is associated with lower rates of LGA, neonatal hypoglycemia and NICU admissions, at the expense of increased prevalence in our population. The ongoing study will include a cost-benefit evaluation to assess if improved outcomes overbalance the increased prevalence inherent to lower diagnostic criteria.

**Diabetes Mellitus and Glucose Metabolism**

**TYPE 1 DIABETES MELLITUS**

**Type 1 Diabetes Diagnosed in an 83 Year Old Man After Nivolumab Therapy**

Marina Joseph, Resident Physician, Madhura Borikar, MD, Deborah I. Bursey, MD.

1University of Arkansas for Medical Sciences, LITTLE ROCK, AR, USA, 2John McClellan Veterans Hospital, Little Rock, AR, USA.

**SAT-686**

**Abstract**

**TITLE**

Type 1 diabetes diagnosed in an 83 year old man after Nivolumab therapy

**BACKGROUND**

Immune checkpoint inhibitors are increasingly being used for a variety of cancers and are a promising treatment option. Immune related adverse effects are their major side effects, most common being hypophysitis and hypothyroidism. While diabetes and adrenalitis have only been rarely reported, these too are becoming more common. We present a case of type 1 diabetes associated with Nivolumab therapy diagnosed in an 83-year-old man.

**CASE**

An 83 year old male with past medical history of emphysema, coronary artery disease, hypertension, non-small cell lung cancer treated with lobectomy, hepatitis C cirrhosis with hepatocellular carcinoma with metastasis to lungs, who completed 10 cycles of Nivolumab presented to our ICU for diabetic ketoacidosis. He did not have a history of diabetes. No family history of diabetes was reported. His C-peptide was found to be low at 0.61. Insulin antibody, Islet cell antibody, Zinc transporter antibody and GAD antibodies were negative. He was discharged on basal bolus insulin regimen. He is being followed in our endocrinology clinic with complaints of polyuria, polydipsia and a weight loss of 10 pounds over the last one week. Lab work showed a blood glucose of 743 with an anion gap of 18 and bicarb of 18. B-hydroxy butyrate was 3.19. He was admitted to our ICU for diabetic ketoacidosis. He did not have a history of diabetes mellitus. No family history of diabetes was reported. His Hemoglobin A1c was found to be 10.1. He had normal blood sugars before starting Nivolumab therapy. His C-peptide was found to be low at 0.61. Insulin antibody, Islet cell antibody, Zinc transporter antibody and GAD antibodies were negative. He was discharged on basal bolus insulin regimen. He is being followed in our endocrinology clinic and continues to be insulin dependent.

**CONCLUSION**

Nivolumab is PD-1 (programmed cell death) inhibitor, which is used as cancer immunotherapy in multiple advanced cancers including hepatocellular carcinoma. Clinically significant endocrinopathies are documented in <5% of patients treated with PD-1 inhibitors. The cause of Diabetes by PD-1 inhibitors is not well defined but believed to be caused by destruction of pancreatic beta cells due to inhibition of autoimmunity by autoreactive T cells. Literature review showed only 42 published cases of PD-1 inhibitor induced type 1 diabetes. Average age at presentation was 62 years and about 69% patients were in DKA at diagnosis. In a recently published study involving 1163 patients who received PD-1 inhibitors, only 21 cases of diabetes were identified, 12 of those were with new onset DM and only 1 case was due to Nivolumab use.

Since this type of endocrinopathy is mainly reported in case reports, we will need more research for further understanding of the pathology so that we can keep a watch out for this adverse effect and prevent life-threatening complications.
Diabetes Mellitus and Glucose Metabolism

TYPE 1 DIABETES MELLITUS

A Case of Opdivo Induced Type 1 Diabetes
Jessica Lucier, MD, Laila Tabatabai, MD.
Houston Methodist Hospital, Houston, TX, USA.

SAT-677

Background: Opdivo, or Nivolumab is an immunotherapy medication that works as a checkpoint inhibitor. It is used in various cancers not amenable to surgery, including lung cancer and metastatic melanoma. Opdivo has proven to be a beneficial treatment, though it is not without complications including thyroid dysfunction, hypophysitis, and autoimmune induced diabetes.

Clinical Case: An 80 year old female with a PMH of type 2 diabetes mellitus, hypertension, hyperlipidemia, and melanoma with metastasis to the bone was evaluated by endocrinology for acute worsening of her type 2 diabetes. The patient had been diagnosed with diabetes 10 years prior. She was well controlled on 22 units of levemir, a humalog sliding scale, and Janumet. At that time it was unclear what precipitated her acute change in glycemic control. Her Janumet was discontinued. She was started on a T-slim insulin pump and Dexcom G6 sensor, with improvement in her glucose control. Her Opdivo treatments were discontinued. She has since developed worsening metastasis in her femur and will be started on Yervoy. Yervoy also carries a risk of further endocrine disorders, including increased risk of hypophysitis.

Discussion: Acute changes in glycemic control warrants further investigation to determine the underlying precipitating factor. The most common complications of Opdivo are rashes and fatigue, though endocrinopathies have been noted in several patients. It was crucial in this patient to identify this side effect of Opdivo, as it helped to prevent further episodes of DKA. Careful review of recent medication changes helped identify this uncommon complication of Opdivo and prompted a timely change in her diabetes care regimen.

Bone and Mineral Metabolism

BONE AND MINERAL CASE REPORTS I

Familial Hyperparathyroidism - Due to a Rare Genetic Mutation
Adeyinka Taiwo, MBBS1, Joseph Stephen Dillon, MD2, Jennifer Stinson, MS CGC1.
1University of Iowa Hospitals and Clinics, Iowa City, IA, USA,
2University of Iowa, Iowa City, IA, USA.

SAT-353

Introduction: Hyperparathyroidism occurs most commonly in middle age patients, predominantly in women. It can be caused by parathyroid adenoma, hyperplasia or parathyroid carcinoma. Genetic predisposition can be found in about 10% of primary hyperparathyroidism due to certain gene mutations. This case emphasizes the importance of taking a detailed family history when patients present with hyperparathyroidism at a young age, so that familial hyperparathyroidism, if present, can be detected and relatives screened.

Clinical case: A 26 y.o. male presented with symptoms of fatigue and polydipsia for several years. He was noted to have a serum calcium of 12.4 mg/dL (8.5–10.5), with parathyroid hormone of 213 pg/ml (15–65). He denied any history of kidney stones, fractures and no palpable neck masses. The patient’s family history was significant for his paternal half-sister who had parathyroidectomy for hyperparathyroidism at 20yrs old and paternal grandmother died of parathyroid cancer in her 50s. The patient’s father died of pancreatic cancer at 41yrs old. A neck ultrasound revealed a mass posterior to the left inferior thyroid. A Sestamibi parathyroid scan revealed a parathyroid adenoma at the postero-inferior aspect of the left hemithyroid. Labs for free metanephrines and normetanephrine, prolactin and gastrin levels were all normal.

Due to his young age and the possibility of having familial hyperparathyroidism, he underwent bilateral neck exploration and parathyroidectomy, with removal of his left inferior, right superior, left superior parathyroid glands and left upper thymus. Surgical pathology revealed, hypercellular parathyroid tissue. Post operatively, his calcium and vitamin D remained within the normal range.