Distant metastasis of follicular thyroid cancer to the bone has been well documented. However, spinal cord compression as the initial presentation of metastatic follicular thyroid cancer without any thyroid symptoms is relatively rare. Here we discuss such a case. A 78-year-old female with history of HTN and melanoma presented to the ED with a 1-month history of middle back pain that progressed to lower extremity weakness, numbness, and inability to ambulate. MRI showed a T7 vertebral mass with cord compression and edema. Metastatic work up was unremarkable except for incidental bilateral thyroid nodules, the largest on the right lobe, at 1.6 cm, with peripheral calcifications. The patient underwent T6-T7 laminectomy with vertebral decompression, partial colpectomy, and T4-T10 fusion. Pathology of the thoracic vertebral mass was positive for follicular carcinoma. The patient denied shortness of breath, dysphagia, hoarseness, or neck tenderness. She had no personal history of hyperthyroidism or hypothyroidism, or radiation exposure. She also did not have any family history of thyroid cancer. Laboratory work up was significant for TSH of 3.71 mIU/mL, FT4 1.56 ng/dl, thyroglobulin (Tg) 6940 ng/mL, and Free T3 1.56 ng/dL, with very similar morphological features to the epidural lesion. In a subgroup analysis, MEN1-related NETs, DNETs and gastric NETs had distinct methylation signatures, respectively, with complete separation by PCA and unsupervised hierarchical clustering. Furthermore, we found CpG hypermethylation in the APC (adenomatous polyposis coli) gene, specifically in the 1A promoter, with higher methylation levels in gastrics- and DNETs vs. SINETs, PNETs and NETs of unknown primary (p < 0.001 for all comparisons).

Conclusion
Various primary NET sites and genetically predisposed MEN1-related NETs have distinct DNA CpG methylation signatures. The methylene signatures identified in this study may be useful for non-invasive molecular characterization of NETs, through DNA methylation profiling of biopsy samples or circulating tumor DNA.
Diabetes Mellitus and Glucose Metabolism

TYPE 1 DIABETES MELLITUS

Partial Beta-Cell Destruction: An Atypical Case of Immune Checkpoint Inhibitor Diabetes Mellitus

Zoe Quandt, MD, MS\(^1\), Katy K. Tsai, MD\(^1\), Victoria C. Hsiao, MD, FHD\(^2\).

\(^1\)UCSF, San Francisco, CA, USA, \(^2\)University of California, San Francisco, CA, USA.

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Background: Autoimmune diabetes mellitus (CPI-DM) caused by immune checkpoint inhibitors (CPIs) is rare—occurring in approximately one percent of patients exposed to this form of cancer immunotherapy. Typically, this immune related adverse event occurs after treatment with PD-1/PD-L1 inhibitors. It is characterized by abrupt insulinopenia leading to acute hyperglycemia. Beta cell autoantibodies are positive in approximately half the cases. DKA is common at the time of diagnosis. Recovery of beta cell function has been reported in only two case reports. In one case, spontaneous resolution occurred following cessation of CPI therapy and in the other the patient was treated with infliximab for concurrent inflammatory arthritis prior to resolution of CPI-DM.

Clinical Case: A 50-year-old woman was started on adjuvant pembrolizumab for stage IIIC melanoma following surgery. She had no prior history of diabetes mellitus, thyroid disease, or other autoimmune disease. Pre-infusion random blood glucose (RBG) were 84 - 105 mg/dL. After 36 weeks, she developed hypothyroidism (TSH 17.5 (0.5-4.1 mIU/L), FT4 6 (10-18 ng/dL)) and started levothyroxine. Pembrolizumab was continued. For nine weeks following her diagnosis with CPI-hypothyroidism, her pre-infusion RBG ranged from 102-133. At 45 weeks (15 cycles) after initiating pembrolizumab, her RBG was 260. She was not on glucocorticoids and had no other signs of inflammation or stress. Pembrolizumab was continued. Just prior to her 17th cycle, 48 weeks after initiating adjuvant pembrolizumab, her RBG was 482 with a normal anion gap and HCO3, and her A1c was 8.9%. Her last dose of pembrolizumab was held. She started metformin and lepraglutide. In just three weeks, a random c-peptide was inadequate at 1.7 (0.8-3.5 ng/mL) with a recent RBG of 220 and A1c of 10.3%, showing the acuity and extremity of her hyperglycemia. Over the course of the year, she has achieved excellent glucose control (A1c 6.3-7.1) on this regimen with preservation of insulin production (c-peptides 1.4-1.8 with matched RBG 92-129). She never required insulin. Her beta cell autoantibodies are negative.

Clinical Lessons: This is a case of CPI-DM in which the patient did not have complete loss of beta-cell function. The acuity of her hyperglycemia is not consistent with new onset type 2 diabetes. At diagnosis, her c-peptide was inadequate suggesting insufficient insulin production rather than insulin resistance. Therefore, her hyperglycemia is more consistent with CPI-DM than type 2 diabetes. Atypically, she did not progress to fulminant beta cell failure, which could have been due to cessation of pembrolizumab (which is not unique to this case), initiation of lepraglutide and metformin, or other unknown immunologic responses that inhibited full beta cell loss. This case raises the possibility of preventing fully insulin dependent CPI-DM if hyperglycemia is caught and treated early.

Adrenal

ADRENAL CASE REPORTS I

A Case of Metastatic Pheochromocytoma Associated with Beckwith-Wiedemann Syndrome

Sindhura Ravindra, MD, Ulil Jagdeesh, MD.

University of Pittsburgh, Pittsburgh, PA, USA.

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Introduction: Beckwith-Wiedemann Syndrome (BWS) is an autosomal dominant disorder of chromosome 11p15 that results in increased IGF-2 and CDK1NC. This leads to excessive cell proliferation and tumor formation. The following highlights a case of metastatic pheochromocytoma in a patient with BWS.

Clinical Case: A 30-year-old male presented with sudden onset blury vision without any associated complaints. His past medical history was significant for BWS. His family history was negative for uncontrolled hypertension, sudden death, thyroid cancer or hyperparathyroidism. Physical examination was notable for an elevated systolic blood pressure of 200/160 mm of hg and fundoscopy revealed features of hypertensive emergency. Laboratory investigations revealed an elevated plasma normetanephrine [10445 pg/ml (normal: <148)], metanephrine [93 pg/ml (normal: <57)], total metanephrine [10538 pg/ml (normal: <205)], epinephrine [134 pg/ml (normal:=50)], norepinephrine [23526 pg/ml (normal: 112-658)], total catecholamine level [23660 (normal: 123-671pg/ml)] and dopamine [405 pg/ml (normal:<30)] levels. His PTH, corrected serum calcium, gastrin, insulin, carcinoembryonic antigen, calcitonin levels and basal metabolic panel were all normal. MRI of the abdomen demonstrated bilateral adrenal nodules with a large mass encasing the celiac axis along with evidence of hepatic lesions. I-123 MIBG scan showed mild radioactive tracer uptake in the adrenal nodules and mass near the celiac axis but not in the hepatic lesions. PET scan confirmed MRI findings and was negative for any evidence of malignancy in the chest, pelvis and skeleton. MRI of the brain was negative for metastasis as well as pituitary abnormalities. Ultrasound-guided liver biopsy was positive for malignant cells that stained positive for chromogranin and synaptophysin confirming the diagnosis of metastatic pheochromocytoma. He was treated with phenoxybenzamine, diltiazem and lisinopril. He underwent cycles of cyclophosphamide, vincristine and dacarbazine. Genetic testing revealed a variant in SDHD gene which was of uncertain significance. Repeat biochemical testing on follow up after a year and a half showed a decreased plasma normetanephrine [487pg/ml] and metanephrine...