with the presence of VF (OR=0.3 (95% CI of 0.2-0.5), P<0.01).
Conclusion: Substantial number of children encounter VF
after ALL treatment is completed and the presence of VF
might be associated with compromised auxological state,
prominent height decline and IGF-1 deficiency.

Diabetes Mellitus and Glucose
Metabolism

TYPE 1 DIABETES MELLITUS
Partial Beta-Cell Destruction: An Atypical Case of
Immune Checkpoint Inhibitor Diabetes Mellitus
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SAT-667
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 im-
mune 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 cessa-
tion 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 ad-
 juvant 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 µg/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 ad-
 juvant 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
liraglutide. 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 reg-
imen with preservation of insulin production (c-peptides
1.4-1.8 with matched RBG 92-129). She never required in-
sulin. 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 liraglutide 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 hypergly-
cemia is caught and treated early.

Adrenal

ADRENAL CASE REPORTS I
A Case of Metastatic Pheochromocytoma Associated
with Beckwith-Wiedemann Syndrome
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SAT-231
Introduction:
Beckwith-Wiedemann Syndrome (BWS) is an autosomal
dominant disorder of chromosome 11p15 that results
in increased IGF-2 and CDK1NC. This leads to exces-
 sive cell proliferation and tumor formation. The following
highlights a case of metastatic pheochromocytoma in a pa-
tient with BWS.
Clinical Case:
A 30-year-old male presented with sudden onset blurry vi-
sion 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 exami-
nation was notable for an elevated systolic blood pressure
of 200/160 mm of hg and fundoscopy revealed features of
hypertensive emergency. Laboratory investigations re-
vealed 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, carcionembryonic 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 evi-
dence of hepatic lesions. I-123 MIBG scan showed mild ra-
dioactive 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

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levels [110 pg/ml] in comparison to his levels on presentation. Repeat imaging revealed a decrease in tumor burden including bilateral adrenal nodules, celiac axis mass and hepatic metastases.

Conclusion:
This is an unusual case of malignant pheochromocytoma in the absence of SDHB mutation in a patient with BWS. Genetic causes in these patients are yet to be determined. However, genes H19 and KCNQ1OT1 have been implicated in addition to IGF-2 and CDK1NC

**Thyroid**

**THYROID CANCER CASE REPORTS I**

**A Retrospective Diagnosis of Malignant Struma Ovarii After Discovery of Pulmonary Metastases**
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SUN-483
**Background:** Malignant struma ovarii is a rare ovarian tumor that is histologically identical to differentiated thyroid carcinoma.1 We present a case of a struma ovarii that was recognized as being malignant only after the discovery of pulmonary metastases.

**Clinical Case:** A 29 year old female presented to the hospital with acute right lower abdominal pain, suspicious for ovarian torsion. She underwent urgent right salpingoopherectomy and pathology demonstrated a mature cystic teratoma with benign struma ovarii. Two years later, a CT of the abdomen incidentally revealed bilateral pulmonary nodules. Review of the imaging showed that these pulmonary nodules were also present two years prior, and had since become larger. Video-assisted thoracoscopic surgery was performed and lung biopsy was positive for well-differentiated thyroid carcinoma. The patient then underwent total thyroidectomy which revealed a 0.3 x 0.3 cm infiltrative papillary thyroid cancer, follicular variant, without lymphovascular invasion. Thyroglobulin level decreased from 169 ng/mL pre-operatively to 80 ng/mL post-operatively, but then continued to be variable ranging from 56 to 252 ng/mL (1.6-50 ng/mL). Thyroglobulin antibodies remained negative.

Pathology from right ovary was re-reviewed at a second institution and found to be consistent with highly differentiated thyroid carcinoma with characteristic nuclear features of papillary thyroid carcinoma. A diagnostic whole body I-131 scan showed uptake within the thyroid bed, bilateral lung nodules, left distal thigh and right mid thigh. These thigh lesions were not visualized on lower extremity ultrasound. After dosimetry was performed, the patient received radioactive iodine-131 200 mCi. Post-therapy scan six days later demonstrated uptake in the thyroid bed, bilateral lungs and bilateral thighs. About five months later, thyroglobulin level had decreased to 0.4 ng/mL with a suppressed TSH. A repeat CT chest demonstrated that the lung nodules had all decreased in size, largest from 0.5 cm to 0.3 cm.

**Conclusion:** Careful examination of struma ovarii pathology should be performed to evaluate for malignant features since benign appearing histology can present diagnostic difficulty.2 In this case, thyroglobulin level was lower than reported in previous cases; however, sites of metastases were responsive to radioactive iodine therapy indicating well differentiated disease and a favorable prognosis.

**References:** 1. Goffredo P, Sawka AM, Pura J, Adam MA, Roman SA, Sosa JA. Malignant Struma Ovarii: A Population-Level Analysis of a Large Series of 68 Patients. Thyroid. 2015;25(2): 211-216. 2. Roth LM, Miller AW, Talerman A. Typical Thyroid-Type Carcinoma Arising in Struma Ovarii: A Report of 4 Cases and Review of Literature. Int J Gynecol Pathol. 2008;27(4): 496-506.

**Thyroid**

**THYROID DISORDERS CASE REPORTS III**

**Thyroid Abscess in a Healthy 22-Year Old Female**
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MON-485
**Background:** Thyroid abscess is a rare pathology, exhibiting an incidence of less than 1% of all thyroid diseases. The thyroid is highly resilient against infections. Those who do experience thyroid abscesses are commonly immunocompromised. We illustrate a case of a thyroid abscess in a young, healthy patient.

**Clinical Case:** A previously healthy 22-year-old woman presented to the emergency department complaining of a sore throat, fever, nausea, and body aches. On exam, the patient was febrile, but had no obvious cervical lymphadopathy, masses, or oropharyngeal lesions/growths. Initial labs showed leukocytosis to 13.5 k/ul with left-shift. The patient was diagnosed with acute pharyngitis, and was discharged on oral steroids and antibiotics for an incidental urinary tract infection.

After some improvement, the patient returned to the ED 14 days later with a worsening odynophagia, dysphagia, and hematemesis. The patient was afebrile, but had neck swelling and possible thyromegaly. Lab results showed leukocytosis to 17.3 k/ul, and CT of the neck identified a 3.1cm x 3.3cm x 4.4cm heterogeneous cystic/solid mass that nearly completely replaced the normal right thyroid lobe parenchyma and extended to the isthmus. Right-sided lymphadenopathy was also present. Initial evaluation suggested thyroid carcinoma. The patient was re-initiated on steroids due previous improvement, and was referred to a tertiary academic medical center for biopsy and further evaluation. 2 days later, the patient returned to the ED for worsening symptoms. However, she was discharged to home with no further management.

At her endocrine surgery consultation visit, the patient reported worsening pain, inability to move her neck, inability to eat or drink, inability to lie flat, and new-onset sialorrhea and voice changes. A bedside ultrasound was performed with findings suggestive of an abscess. An in-office fine-needle aspiration produced purulent fluid, which relieved some of