CONCLUSION
Early course of AMTT can rarely present as AST, both being progressive and fatal diseases. Our case had a typical presentation of AST, causing delay in diagnosis of his very aggressive form of thyroid cancer. Gross pathology showed predominantly hemorrhagic areas in the tumor with central necrosis, which was likely the cause of acute mass with systemic signs suspicious for infection. One should consider an alternative diagnosis in suspected cases of AST in the absence of positive cultures or lack of improvement with parenteral antibiotics. It is imperative to make a diagnosis without any delay as prognosis for either condition depends on prompt recognition and treatment.

Thyroid

THYROID CANCER CASE REPORTS I

An Unusual Case of Poorly Differentiated Thyroid Carcinoma with an Excellent Prognosis

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SUN-474

Background: Poorly differentiated thyroid carcinoma (PDTC) is a rare and aggressive subtype with morphological/behavioral features between differentiated thyroid carcinoma (DTC) and anaplastic thyroid carcinoma (ATC).
Clinical case: A 43-year-old female presented with 3 cm right thyroid mass noted on US neck. FNA biopsy showed undifferentiated carcinoma, large cell type. Additional immune-stains were suggestive of ATC. Pre-surgery non-stimulated thyroglobulin (NSTG) was 311 (RR 0–55 ng/dl). Pathology post-total thyroidectomy with bilateral level VI lymph node dissection showed a 3.2 x 2.5 x 2.5 cm carcinoma with vascular and capsular invasion. Most of the mass consisted of very atypical pleomorphic cells, mitosis was difficult to find. The tumor did not show the widely invasive-destructive pattern commonly seen in ATC. An adjacent differentiated component showed predominantly follicular pattern and was described as dedifferentiated follicular carcinoma. All lymph nodes were negative for metastatic disease. Post-surgery NSTG was <0.2 (RR <0.1ng/ml as athyreotic), stimulated TG was 2.22 with negative TG antibodies. Four months later, she received 193.5 mCi radioactive iodine (RAI) therapy. The post-therapy scan showed no Iodine-131 avid uptake in neck or distant metastasis. Neck imaging and TG levels done periodically showed no structural or biochemical evidence of recurrence. Currently the patient is cancer-free for 14 years since diagnosis with no need for additional therapies.
Discussion: PDTC accounts for 1–15% of all thyroid cancers. Although PDTC is rare, it is a clinically significant histological diagnosis as it represents the main cause of death from non-anaplastic follicular cell-derived thyroid carcinoma. The Turin proposal published in 2007 suggested three criteria for the diagnosis of PDTC which included the pattern of growth and high-grade features. PDTC presents more frequently with locally invasive extra-thyroidal disease, metastasis to regional lymph nodes and distant organs compared to DTC. Despite the capacity to have RAI uptake, there has been no evidence of significant improvement in survival due to tumor heterogeneity in differentiation. Recent data suggest that age more than 45 years, tumor size more than 4cm, extra-thyroidal extension, higher pathological T stage, positive margins, and distant metastasis predict worse prognosis.
Conclusion: Our patient showed an excellent response to therapy in spite of having PDTC with positive margins. We hypothesize that this could be likely due to young age at the time of diagnosis, early detection of tumor while it was localized in the thyroid without distant metastasis as well as heterogeneity in the tumor with differentiated cells that are responsive to RAI. We conclude that with early detection, timely surgery, and adjuvant therapy, excellent prognosis can be achieved in patients with PDTC.

Adrenal

ADRENAL CASE REPORTS II

Pseudo-Cushing Syndrome Secondary to Malnutrition and Gluco-Toxicity Mimicking Type 1 Diabetes Mellitus

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SUN-205

Background: Pseudo-Cushing Syndrome (PCS) is an under-recognized clinical entity that is a reversible consequence of alterations in cortisol production. We present a case of a patient with presumed type 1 Diabetes Mellitus (DM) who was found to have PCS secondary to malnutrition. Once the nutritional status normalized, the patient's glycemic control remarkably improved and became well-controlled on metformin alone.
Clinical case: A 54-year-old female with poorly controlled insulin-dependent DM for 10 years was referred for concern for adrenal insufficiency after an ACTH came back elevated in the setting of intractable nausea, vomiting and considerable weight loss over 1 year. Prior HbA1c was 16.2% (RR 4.4–6.7). On exam her vitals were normal, body mass index (BMI) was 15 kg/m^2. Workup confirmed an elevated ACTH of 100 pg/ml (RR 6–50 pg/ml), however, random PM cortisol was unexpectedly elevated at 26.58 ug/dL (RR 4.46 – 22.7). 8 AM labs for ACTH and cortisol were similarly elevated at 91 pg/ml and 28.33 ug/dl, respectively. She had no evidence of classic Cushingoid features. Subsequent low dose dexamethasone suppression test and 24-hour urine free cortisol were negative. Over 18 months, with optimization of her insulin therapy, BMI improved to 19 kg/m^2, ACTH and cortisol started to downtrend spontaneously. After 30 months, her BMI improved to 20 kg/m^2. Repeat blood work showed A1C 6.5%, ACTH and cortisol completely normalized to 42 pg/dl and 8 ug/dL, respectively. After being adherent to insulin for a few years, her gluco-toxicity state resolved. A month prior to following up, she self-discontinued insulin due to hypoglycemia but continued on metformin. Currently she continues to remain off insulin.
Discussion: PCS is a challenging diagnosis to recognize and differentiate from Cushing Disease (CD) especially due to overlap in biochemical profile. It is important to be aware of

this clinical condition to avoid misdiagnosis, delay in treatment or over-treatment. Common etiologies causing PCS include depression, chronic alcoholism, obesity, physical stress, malnutrition, eating disorders, uncontrolled DM, obstructive sleep apnea. PCS occurs due to chronic activation of the hypothalamic-pituitary-adrenal axis, it is usually mild and resolves with treatment of underlying etiology. In our case, first-line screening tests could differentiate between PCS and CD hence she did not require late-night salivary cortisol testing or corticotropic-releasing hormone testing.
Conclusion: In our patient, PCS occurred secondary to malnutrition and severe gluco-toxicity which mimicked insulin-dependent type 1 DM. Interestingly, once her nutritional status and insulin compliance improved, cortisol levels normalized, gluco-toxicity state resolved and she no longer required exogenous insulin therapy.

Neuroendocrinology and Pituitary

PITUITARY TUMORS I

3D Mapping of the Human Growth Hormone Locus Identifies Putative Regulatory Hubs for Genes Involved in Cellular Signalling and Cancer-Related Pathways

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SAT-303

Growth hormone (GH) is a peptide hormone predominantly produced in the pituitary that is crucial for normal growth and metabolism. Downstream effects of GH are mediated through binding to the GH receptor (GHR) and consequent activation of key signalling cascades including JAK-STAT, MAPK, PI3K-Akt and mTOR. The *GH* locus is comprised of five evolutionarily related genes under the control of an upstream locus control region which coordinates tissue-specific expression of these genes in the pituitary and placenta (1). Compromised GH signalling and genetic variation in these genes has been implicated in various disorders including cancer. We hypothesised that polymorphisms which occur within the *GH* locus have the potential to impact on disease phenotypes by altering or disrupting gene regulation. We used the CoDeS3D (Contextualize Developmental SNPs using 3D Information) algorithm to analyse 529 common single nucleotide polymorphisms (SNPs) across the locus. This algorithm identifies colocalised Hi-C and eQTL associations to determine which SNPs are associated with a change in gene expression at loci that physically interact within the nucleus. We identified 181 common SNPs that interacted with 292 eGenes in 48 different tissues. 145 eGenes were regulated in *trans*. We performed pathway enrichment of identified eGenes and found these to be enriched in GH/GHR-related downstream cellular signalling pathways including MAPK, PI3K-Akt-mTOR, and ErbB signalling. Enrichment was also observed in the Wnt and Hippo signalling pathways. There was also a significant representation of these eGenes in pathways associated with hepatocellular, colorectal, breast and non-small cell lung carcinoma. 33 eQTL SNPs identified in our study were found to be of regulatory importance in a genome-wide Survey of Regulatory Elements (SuRE) reporter screen (2). In addition, 7 eQTL SNPs were located in known enhancer regions. Our data suggests that regions within the *GH* locus form regulatory hubs for multiple genes in *cis* and *trans* (intra and inter-chromosomal), many of which are involved in mediating GH function in normal and pathogenic states.
Reference: (1) Tsai et al. Nucleic Acids Res 2016, 44, 10, 4651 (2) van Arensbergen et al. Nat Genet 2019, 51, 7, 1160.

Cardiovascular Endocrinology

HYPERTRIGLYCERIDEMIA; INFLAMMATION AND MUSCLE METABOLISM IN OBESITY AND WEIGHT LOSS I

Experiences with Hypertriglyceridemic Pancreatitis: A Mini Case Series

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SAT-579

Background: Hypertriglyceridemia (HTG) is a well-established cause of acute pancreatitis (AP) in up to 14% of all cases & up to 56% cases during pregnancy. The triad of HTG, Diabetic ketoacidosis (DKA) and AP is rarely seen posing diagnostic challenges. Early recognition of HTG-induced pancreatitis (HTGP) is important to provide appropriate therapy & prevent recurrence. In this case series, we discuss the diagnostic challenges and clinical features of HTGP. ***Clinical cases:*** Our first patient was a 65-year-old male with a history of hypertension who presented to the ER with abdominal pain and new-onset pruritic skin rash after a heavy meal. His exam and labs were notable for a diffuse papular rash on his back, triglycerides (TG) of 7073mg/dL (normal: <150mg/dL). The rash improved with the resolution of HTG. Our second patient was a 29-year-old male with a history of alcohol dependence who was found to have AP complicated by ARDS requiring intubation. Further testing revealed that his TG was 12,862mg/dL & his sodium (Na) was 102mEq/L. Although HTG was known to cause pseudohyponatremia, it was a diagnostic challenge to estimate the true Na level. In a third scenario, a 28-year-old female with a history of T2DM on Insulin presented with nausea & abdominal pain. Labs were suggestive of DKA and lipase was normal. CT abdomen showed changes consistent with AP. The TG level that was later added on was elevated to 4413mg/dL. She was treated with insulin that improved her TG level. ***Discussion:*** We present three cases of hypertriglyceridemic pancreatitis. While the presentation can be similar to other causes of acute pancreatitis (AP), there are factors in the diagnosis and management of HTGP that are important to understand. Occasionally, physical exam findings can be suggestive of underlying HTG. In the first scenario, our patient presented with eruptive xanthomas - a sudden eruption of crops of papules that can be pruritic. They are highly suggestive of HTG, often associated with serum TG levels > 1500mg/dL. Our second patient presented with pseudohyponatremia.