in favor of iv insulin and fluid resuscitation, with admission to the medical intensive care unit. Further lab testing demonstrated low C-peptide levels and positive IA-2 and GAD-65 antibodies, confirming autoimmune diabetes. Endoscopic biopsy was also consistent with autoimmune colitis, and TSH one month after discharge was 123.70 (0.30-5.00 mcIU/mL) with free T4 < 0.1 (0.6-1.6 ng/dL).

Despite early discontinuation of anti-PD1 therapy the melanoma has remained in remission for three years, suggesting a sustained immune response. He continues to require insulin and thyroid hormone replacement, though the autoimmune colitis has resolved.

Conclusion: This case demonstrates the overall benefit of immune checkpoint inhibitor therapy in the treatment of metastatic melanoma, while highlighting a potentially lethal therapy complication with the concurrent onset of multiple autoimmune processes affecting separate organ systems. Increased awareness of the potential for DKA in patients not previously diagnosed with diabetes is needed to avoid delays in care and improve outcomes. This case also suggests a potential benefit to integrating routine blood glucose monitoring in immune checkpoint inhibitor treatment protocols, the utility of which should be further investigated.

Neuroendocrinology and Pituitary PITUITARY TUMORS II

The Prevalence of Acromegaly in the Sleep Apnoea Clinic

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MON-302

The Prevalence of Acromegaly in the Sleep Apnoea Clinic **Introduction**: The prevalence of acromegaly in the general population ranges 4-14/100,000. 45-80% of acromegaly patients have obstructive sleep apnoea (OSA). The OSA population might represent a target group for earlier detection of acromegaly, thereby reducing associated long-term morbidity.

Methods: Patients attending the sleep service (11/2014-04/2018) were recruited in a prospective multicentre cohort study. All had serum IGF-1 measurement and completed a screening questionnaire for five key symptoms associated with acromegaly. Those with raised age-specific IGF-1 underwent further biochemical assessment to investigate for acromegaly.

Results: 1080 participants (73% male, mean age 55.6±12.0yrs) with confirmed OSA were recruited across two sites. Forty-three patients (4%) reported at least 4/5 acromegaly-related symptoms. There was no correlation between serum IGF-1 and symptom score. Sixty-one patients (5.7%) had elevated IGF-1 level on initial assessment. Fifty-one had repeat IGF-1 testing, while one had

growth hormone measurement of $<1\mu g/L$. Nine patients were lost to follow-up, including one death.

Of the repeat IGF-1 tests, results were normal in 24 cases and no further investigation was undertaken. Repeat IGF-1 results were unavailable in 3 cases. In the remaining 24 patients with persistently raised IGF-1, 11 had GH <1 μ g/L, suggesting that acromegaly was unlikely. The remainder (n=13), as well as the 3 individuals with unavailable IGF-1 results, had an oral glucose tolerance test. One patient (BMI of 23.7kg/m²) was diagnosed with acromegaly, was diagnosed with severe OSA and reported 4/5 acromegaly-related symptoms during screening.

Conclusion: Our study identified a single case of acromegaly within the OSA population that may represent a higher prevalence than in the background population, however is based on a single case. As a consequence of the significant number of patients with elevated serum IGF-1 measurements requiring further investigation, IGF-I is not currently a cost-effective screening tool for early detection of acromegaly in OSA patients.

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Thyroid

HPT-AXIS AND THYROID HORMONE ACTION

Reduced Cholesterol Absorption and Synthesis Markers in Patients with Hyperthyroidism Due to Graves' Disease

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SAT-456

Background: Thyroid hormones have been reported to promote cell-surface expression of low-density lipoprotein receptor (LDL-R), and also increase mRNA expression of HMG-CoA reductase at the same time. Since LDL cholesterol (LDL-C) uptake via LDL-R is relatively superior to cholesterol synthesis in hyperthyroidism, plasma LDL-C levels can be lower as compared to euthyroid state. Conversely, hypothyroidism can increase plasma LDL-C levels because cholesterol absorption via Niemann-Pick C1-like 1 has been suggested to increase in hypothyroidism. However, there have been no reports about changes of cholesterol absorption and synthesis markers by the treatment of hyperthyroidism in patients with Graves' disease. Patients and method: We collected plasma samples from patients with hyperthyroidism, who were diagnosed as Graves' disease (n=17, M/F: 4/13, age: 24-70 years old). Thyroid hormones, general lipid profiles (Total cholesterol: TC, LDL-C, high-density lipoprotein cholesterol: HDL-C and triglyceride: TG), apolipoproteins, markers of cholesterol synthesis (lathosterol) and absorption (campesterol, sitosterol, cholestanol), lipoprotein lipase (LPL), and proprotein convertase subtilisin/kexin type 9 (PCSK9) were analyzed before treatment, and at euthyroid state (eu), 3 and 6 months after attaining euthyroid state (eu-3M and eu-6M). Result: It took 159.2±108.6 days to attain euthyroid state by the thiamazole treatment. TC, LDL-C and HDL-C levels were increased at eu (TC, 144.5±26.7 to 225.0±61.6; LDL-C, 77.8±20.9 to 138.9±43.9; HDL-C, 49.7±12.6 to 67.9±20.0 mg/dL: P<0.0001 vs before treatment, respectively). Such changes remained at eu-3M and eu-6M. TG was not changed at eu, but significantly increased at eu-6M (85.0±49.1 to 113.7±60.8 mg/dL, P=0.02). Cholesterol absorption markers were increased at eu, eu-3M and eu-6M (e.g. campesterol, 2.6 ± 1.2 to 4.9 ± 2.3 ; sitosterol, 1.5 ± 0.6 to 2.9 ± 1.4 ; cholestanol, 1.9 ± 0.6 to 3.2 ± 1.1 µg/mL: P<0.0001, eu vs before treatment, respectively). Cholesterol synthesis marker was increased at eu, eu-3M and eu-6M (e.g. lathosterol, 1.8±0.7 to 2.3±0.9 μg/mL: P=0.005, eu vs before treatment). Both LPL and PCSK9 were also increased at eu, eu-3M and eu-6M. Conclusion: These data suggest that both cholesterol absorption and synthesis are downregulated in patients with hyperthyroidism due to Graves' disease and can be restored by attaining euthyroid state. In turn, LDL-C and TG levels should be carefully monitored during the treatment of Graves' disease because hyperlipidemia could emerge in euthyroid state.

Thyroid

THYROID NEOPLASIA AND CANCER

Clinical-Pathological and Molecular Prognostic Markers in Aggressive and Poorly Differentiated Thyroid Cancers; A Tertiary-Center Experience Suhaib Radi, MD, Sabin Filimon, MD, Michael Tamilia, MD. McGill Univ, Montreal, QC, Canada.

MON-502

Background:

Aggressive variants of papillary thyroid cancer (AV-PTC) and poorly differentiated thyroid cancers (PDTC) are 2 malignancies that lie in between the well-differentiated and the undifferentiated anaplastic cancers. While management of those well-differentiated cancers is established in the literature, that of AV-PTC and PDTC is less clear as they behave different to their more benign counterparts. The aim of this study is to describe the clinico-pathologic characteristics and genotypic background of AV-PTC and PDTC and to assess their prognostic value.

Methods:

The charts of all patients with thyroid cancer in our center for the last 10 years were retrospectively reviewed. Those with AV-PTC and PDTC were selected and included in the analysis. Clinical presentation, pathologic characteristics, molecular markers, specific treatments and clinical outcomes were compared among groups.

Results:

Out of 3244 thyroid cancer charts reviewed, 87 patients met the criteria for AV-PTC (n=45) and PDTC (n=42). Mean age at diagnosis was 48.1 years (SD 17.8), with female predominance (64.4% vs 35.6%). Median duration of follow up was 3 years (0.1-30). Out of the 75 patients with follow up for more than a year, 42.7% had either persistent disease or recurrence (52.6% in AV-PTC and 32.4% in PDTC) and 4.1% died. Presence of vascular invasion was associated with higher rates of persistent or recurrent disease (74.1% in positive vascular invasion vs 20.5% in negative vascular invasion, p < 0.001). Recurrence rate was 0% in patients with Ki67 < 10% and 40% in those >= 10%. There

was no difference in terms of recurrence based on presence of BRAF mutation (33% in BRAF+ & 29% in BRAF-, p=1), or percentage of

aggressive/poorly differentiated tumor involvement (48% in > 30% involvement vs 28% in < 30%, p = 0.132).

Discussion and conclusion:

The prevalence of AV-PTC and PDTC in this cohort was low at 1.3% each, and the rate of patients with persistent or recurrent disease at 1 year after primary therapy was also similar to that reported (42.7%). The mortality rates, however, in our study is surprisingly lower than that expected elsewhere (4.1%), most likely attributed to a shorter follow up period. Patients with absent vascular invasion were less likely to have persistent or recurrent disease. Those with lower Ki67 (<10%) also had lower relapse rate, although, the p value was > 0.05. It is worth mentioning that even though there were higher rates of recurrence among those with > 30% tumor involvement, it did not reach statistical significance, supporting recent studies stating that even tumor involvement of > 10% can have adverse outcomes. In conclusion, AV-PTC and PDTC are relatively rare but aggressive tumors. Possible prognostic markers that can be used to guide therapy and monitoring include: vascular invasion, extra-thyroidal extension, response to primary therapy and the proliferative index Ki67.

Neuroendocrinology and Pituitary CASE REPORTS IN SECRETORY PITUITARY PATHOLOGIES, THEIR TREATMENTS AND OUTCOMES

Thyrotropin Secreting Pituitary Adenoma Initially Misdiagnosed as Primary Hyperthyroidism in a Taiwanese Man

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SAT-268

Background:TSH (Thyrotropin) secreting pituitary adenoma (TSHoma) account for less than 1% of all causes of hyperthyroidism and 1% of all functioning pituitary tumors. Definite diagnosis and treatment of TSHoma are clinical challenges in practice. Here we report laboratory data, imaging findings, endocrine dynamic test, and treatment outcomes in a 50-year-old Taiwanese man with pituitary plurihormonal adenoma secreting TSH and LH. Clinical case: The patient was initially diagnosed as goiter with primary hyperthyroidism and DM while medical check-up by primary care physician in 2014. He had no significant hyperthyroidism symptoms and signs except goiter and mild palpitation. He received propylthiouracil and Metformin. Two years later, he visited to Endocrinologist's clinic for poor glycemic control. Central hyperthyroidism was diagnosed due to measurable TSH level in the presence