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

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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

of increased serum thyroid hormone level. Moreover sella MRI revealed left sided pituitary lesion. He was referred to Taipei Veteran General Hospital for further management. There was no family history of thyroid disease. Physical examination was not remarkable except diffuse grade 3 goiter and tachycardia (HR 100~115 bpm). Follow up laboratory data showed TSH 4.89; range 0.4~4.0 uIU/ml, free T4: 3.05; range 0.9~1.8 ng/dl, T4: 16.02; range 4.50~12.50 μg/dl, T3: 249; range 58~159 ng/dl, free T3: 8.0; range 2.3~4.3 pg/ml. Two times of TRH stimulation test showed blunted TSH response. Normal limit of thyroid autoantibodies level were found. Thyroid sonography revealed heterogenous echogenicity with increased size and vascularity of both lobes. I-131 uptake was homogenous uptake (94%). Other pituitary hormones level were within normal limit except mild elevation of testosterone 12.69 ng/ml. Sella MRI with contrast showed macroadenoma (size 10x10x7.6 mm) at left pituitary gland. Taken together, he was diagnosed as central hyperthyroidism related to left sided pituitary macroadenoma. Surgery was performed after one year of definite diagnosis due to personal reason. TSH level returned to normal ranges (0.799 uIU/ml) in 1st post operative day. Histologically, the pituitary mass was compatible with plurihormonal adenoma and immunohistochemistry showed positivity for TSH (4+) and LH (3+). Post operative condition was well. Antithyroid agent was discontinued after operation. His
blood glucose became well controlled after operation.
Clinical lessons:A biochemical hallmark of TSHoma is an escape of TSH from the feedback loop that is detectable TSH levels in the presence of increased serum thyroid hormone level. Diagnosis of TSHoma was frequently unrecognized and thus much delayed despite its relatively straightforward. Physician should keep in mind that the importance interpretation of simple laboratory tests to avoid delay diagnosis and unnecessary treatments.

Pediatric Endocrinology

PEDIATRIC PUBERTY, TRANSGENDER HEALTH, AND GENERAL ENDOCRINE

Diversity of Endocrine Function in Patients with CHARGE Association

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SUN-064

Context: CHARGE association consists of congenital malformation of Coloboma, Heart defect, Atresia choanae, Retarded growth and development, Genital hypoplasia, Ear anomalies and/or deafness. It is often caused by *CHD7* gene mutation, which also one of the causative gene for Kallmann syndrome. The endocrine dysfunction in CHARGE association has been reported but not fully understood. Objective: To clarify the mode of growth and frequency of endocrine dysfunction in CHARGE association. Subjective: We investigated the characteristics of growth and puberty, and endocrine function in 23 children (15 males and 8 females, 0~20 years old) with CHARGE association. Results: The birthweight was from -2.74 to +1.14 SDS and the birth length was from -2.86 to +1.10 SDS. 5 children were born small for gestational age. The height below -2SDS in 18 children. GH secretion was evaluated in 11 children with short stature (-9 to -2.3SD) except for one with normal height (-0.3 SD in 6 years old girl); 5 children including one with normal stature were revealed to have GH deficiency. One short girl with GH deficiency previously showed normal GH response to provocation test at 1 year old but has developed to be GH deficient at 7 years old. Gonadotropin-releasing hormone loading tests were performed in 7 males and 3 females. Nine out of 10 children showed hypogonadotropic hypogonadism; one girl showed hypergonadotropic hypogonadism, whose ovaries were undetectable on ultrasound. Human chorionic gonadotrophin (HCG) tests were performed in 6 males with micropenis and/or cryptorchidism. Peak testosterone levels after HCG stimulation were from 0 to 6.99 ng/ml. 4 patients showed peak testosterone levels less than 1 ng/ml. Four boys showed combined gonadotropin deficiency and primary hypogonadism. Conclusions: Our data showed the diversity of endocrine function in children with CHARGE association. GH deficiency can be developed over time. Hypogonadotropic hypogonadism is common, while isolated/combined primary hypogonadism should be taken into consideration in children with CHARGE association.

Bone and Mineral Metabolism

BONE AND MINERAL CASE REPORTS I

Case Series of Ectopic Parathyroid Gland

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SAT-377

The prevalence of mediastinal parathyroid adenoma is unknown. Embryological origin and more extensive aberrant migration of the parathyroid glands result in ectopic glands found in the mediastinum. We report herein 4 cases of ectopic parathyroid adenoma causing primary hyperparathyroidism from three public hospitals in MalaysiaCase 1.A 70 year old lady with underlying diabetes mellitus, hypertension, chronic immune thrombocytopenic purpura and liver cirrhosis presented with incidental asymptomatic hypercalcemia during an admission for pneumonia. Her blood results revealed high corrected calcium of 2.93 mmol/L (2.02-2.60) and a low phosphate of 0.66 (0.81-1.45) mmol/L with an unsuppressed intact parathyroid hormone (iPTH) of 14.56 pmol/L (1.6-6.9). She had an equivocal urinary calcium excretion ratio of 0.01. Her bone mineral density confirmed severe osteoporosis at distal radius and neck of femur with a Tscore of -3.6 and -3.1 respectively. A hyperfunctioning ectopic parathyroid gland was seen in the Technetium Sestamibi scan which corelates with a mediastinal lymphadenopathy on CECT. The largest node measured 1.6 x 1.2 cm. Parathyroid gland was confirmed on HPE of the video-assisted-thoracoscopic surgical (VATS) excision of the mediastinal mass. Intraoperative iPTH