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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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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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