

On average, patients screened positive for 2 (median value) eating behavior traits. Four (5.41%) patients screened positive for five eating behavior traits, the maximum observed in our sample. Biserial correlation analysis showed that satiety was correlated with the FTO gene (Pearson's $r=0.502$, $p<0.001$) and eating disinhibition was negatively correlated with hunger (Pearson's $r=-0.450$, $p=0.034$). Overall, overweight and obese patients had a disproportionately high incidence of eating disinhibition, food desire, and the FTO obese gene. These atypical behaviors can contribute to their difficulty in losing weight. Specific strategies can be discussed with patients around their atypical behaviors with regular follow up appointments by the clinician. Genetic testing can provide important patient education to improve outcomes related to weight management and health outcomes.

Adrenal

ADRENAL CASE REPORTS III

Isolated Hypoaldosteronism Due to Autoimmune Adrenalitis in a Patient With Autoimmune Polyglandular Syndrome.

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BACKGROUND

At the initial presentation of autoimmune adrenal insufficiency, most patients present with hormonal deficiencies from all three layers of adrenal cortex. However, isolated aldosterone deficiency causing a true partial adrenal insufficiency in the setting of autoimmune adrenalitis remains underrecognized.

CASE REPORT

A 67-year old female patient with a known history of diabetes mellitus type 1 since the age of 13 and morphea, presented with progressively worsening symptoms of confusion and hallucinations, fatigue, and loss of appetite over the past 5 years. During this time, she has had frequent and recurrent episodes of mild intermittent hyponatremia with hyperkalemia requiring intravenous fluids and ingested salt tablets, especially when she felt more symptomatic. On her initial evaluation here, she presented with hyponatremia (125 mmol/l, n: 135-145 mmol/l), low osmolality (264 mOsm/kg, n: 275-295 mOsm/kg), and normal potassium level (3.6 mmol/l, n: 3.6-5.2 mmol/l). Further investigations drawn at the same time revealed a low aldosterone (<4 ng/dL), normal renin (5.3 ng/mL/hr, ref 2.9-10.8), normal serum cortisol level (and normal response to Cortrosyn stimulation), though all in the setting of positive antibodies against 21-hydroxylase. Pan-imaging revealed no evidence of malignancy that can be causing ectopic SIADH production. Additional testing showed presence of auto antibodies contributing to pernicious anemia and thyroid disease. Treatment was started with fludrocortisone 0.1 mg tablet daily and she was advised to take the salt tablets only if she has any symptoms. The patient's symptoms have resolved 8 months since this diagnosis, with normalized sodium and potassium levels.

CONCLUSION

Autoimmune primary adrenal insufficiency usually affects all three layers of the adrenal cortex, where patients present with deficiencies in cortisol and aldosterone. Isolated hypoaldosteronism has rarely been reported, however because it can cause life-threatening hyponatremia, it is an important entity to recognize. It is important to work up in such patients as they may be in the initial stages of autoimmune Addison's disease and can progress to developing cortisol deficiency, though the time course to this progression is not well known.

Pediatric Endocrinology

PEDIATRIC SEXUAL DIFFERENTIATION, PUBERTY, AND BONE BIOLOGY

Serum 25-Hydroxyvitamin D Is Not Associated With the Type of Central Precocious Puberty in Girls

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Changes of serum 25OHD levels in girls with different types of central precocious puberty

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[Objective] To evaluate the clinical value of serum 25-hydroxyvitamin D (25OHD) in girls with different types of central precocious puberty (CPP), in order to provide basis for the clinical diagnosis and treatment.

[Methods] 340 CPP girls diagnosed in our hospital from January 2016 to January 2018 were enrolled and retrospectively studied. According to the progression of Tanner stage ≥ 1 during 6 months, bone age(BA) levels were higher than chronological age of more than 1 year. 226 patients were included in the rapidly progressive CPP group (RP-CPP), while 114 patients were included in the slowly progressive CPP group (SP-CPP) as a control. We analyzed the correlation between serum 25OHD levels and the different puberty characteristics (BA, disease course, body mass index (BMI), bone mineral density (BMD), serum LH peak to FSH peak ratio (LHP/FSHP), insulin-like growth factor 1(IGF1)) of two groups. According to sunshine duration, the sampling season was divided into two groups (December to May, June to November), then we compare the correlation between different serum 25OHD levels and season of sampling as well as the different puberty characteristics respectively.

[Results] (1) The mean serum 25OHD levels of CPP girls were 15.89 ± 6.87 ng/ml. The 25OHD levels of 68 (20.0%), 95 (27.9%) and 167 (49.1%) patients were <10, 10-15 and 16-29 ng/mL, respectively. Only 10 (2.9%) patients had normal 25OHD (>30 ng/mL). (2) No significant difference in serum 25OHD levels between RP-CPP group and SP-CPP group ($F=0.809$, $p=0.369$) was found. There is no correlation of BMD and disease course between the two groups ($p>0.1$). Bone age, BMI, LHP/FSHP and IGF1 levels in RP-CPP group were higher than SP-CPP group ($P<0.05$). Logistic regression analysis showed that

BMI, LHP/FSHP and IGF1 were the independent risk factors for CPP (OR 2.690, 1.005, 3.288, respectively). (3) There were significant differences among different serum 25OHD levels as for season, disease course and IGF1 ($p < 0.05$). The correlation with the season was the highest ($r = 0.402$, $p < 0.001$).

[Conclusions] (1) Vitamin D levels are generally insufficient in CPP girls and are not related to different types of CPP. (2) The higher BMI, IGF1, LHP/FSHP levels are, the easier CPP girls will transfer to RP-CPP, but not associated with vitamin D levels. (3) CPP girls suffer from vitamin D deficiency in seasons of winter and spring easier.

[Key words] precocious puberty; vitamin D; female

Pediatric Endocrinology

PEDIATRIC SEXUAL DIFFERENTIATION, PUBERTY, AND BONE BIOLOGY

The Association of Growth Hormone Treatment in Children With Short Stature With Idiopathic Scoliosis

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Objectives: Adolescent idiopathic scoliosis (AIS) is the most common form of scoliosis, which occurs usually in the periods of growth spurts and puberty changes. The spinal curve progression in AIS is related to growth, skeletal maturity and sexual maturity. Growth hormone treatment has been used to improve final adult height by increasing growth velocity in children with short stature. Therefore, AIS is expected to occur more frequently in children treated with growth hormone. The aim of this study was to investigate the relationship between AIS and growth hormone treatment in short children. **Methods:** A total 115 subjects aged 2.1 to 16.9 years who had been treated with growth hormone because of growth hormone deficiency ($n = 83$), idiopathic short stature ($n = 16$) and small for gestational age ($n = 16$) were included. Their medical records were reviewed retrospectively. The scoliosis angle of each subject was measured on the standing frontal radiograph according to the Cobb method. A curve with a Cobb angle of 10 degrees or more is defined scoliosis. Follow up Cobb angle were measured after 1-year of growth hormone treatment. **Results:** Cobb angle has been increased after 1-year growth hormone treatment (6.1 ± 3.1 vs. 6.9 ± 3.4 , $P = 0.024$). The change of Cobb angle was correlated with serum insulin like growth factor-1 level at baseline ($r = 0.274$, $P = 0.003$) and after 1-year of growth hormone treatment ($r = 0.220$, $P = 0.020$). There was no significant correlation between the change of Cobb angle and growth velocity. The prevalence of AIS has been increased after 1-year of growth hormone treatment (11.3% vs. 19.1%, $P = 0.009$). **Conclusion:** Growth hormone treatment in children with short stature seems to be associated with occurring and worsening of AIS.

Adipose Tissue, Appetite, and Obesity

ADIPOSE TISSUE BIOLOGY AND OBESITY II

Nrf2 Represses Obesity-Associated Adipose Tissue Inflammation in Mice

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Obesity is associated with type 2 diabetes, cardiovascular disease and increased incidence of cancer. Chronic inflammation, mainly emanating from adipose tissue, has been proposed to be one of the links between obesity and these pathologies. Thus, identification of new targets against obesity and especially obesity-induced inflammation is needed urgently. Transcription factor Nrf2 (NF-E2-related-factor-2) plays a central role in cytoprotective responses to oxidative and electrophilic stresses and also exerts anti-inflammatory effects in rodent models of inflammation. However, whether activation of Nrf2 signaling pathway influences obesity-associated inflammation in adipose tissue is not well established. To this end, we generated mice with systemic activation of the Nrf2 pathway (*Keap1^{flox/-}*), as well as mouse models with tissue-specific Nrf2 pathway activation: adipocyte-specific (*Fabp4Cre::Keap1^{flox/flox}*) and myeloid cell-specific (*LymCre::Keap1^{flox/flox}*). These mice were exposed to a high-fat diet (HFD) 60% kcal fat regimen for 6-weeks or crossed into the *db/db* background. *Keap1^{flox/-}* mice showed a dramatic decrease of the numbers of F4/80-positive macrophages in white adipose tissue (WAT). Interestingly, both *Fabp4Cre::Keap1^{flox/flox}* and *LymCre::Keap1^{flox/flox}* mice showed suppression of F4/80-positive macrophages in WAT as well, suggesting enhanced Nrf2 signaling in either adipocytes or myeloid cells might contribute to anti-inflammatory effects in WAT under the stress of HFD. Transcript levels of inflammatory markers, especially macrophage *F4/80* and *Cd68* and the chemokine *Ccl2* were decreased in the WAT from *Keap1^{flox/-}* mice on the standard diet and also in the WAT of *Keap1^{flox/-}* mice in the *db/db* background. Pharmacological activation of the Nrf2 pathway by treatment with CDDO-Im also suppressed *Ccl2* expression in WAT of HFD fed mice and *db/db* mice. As CCL2 is a key mediator of macrophage accumulation in adipose tissue, we further studied the potential effect of Nrf2 on the transcriptional regulation of *Ccl2* using 3T3-L1 preadipocyte and RAW264.7 macrophage cell lines. Treatment of both lines with the small molecule inducer of Nrf2, diethyl maleate significantly suppressed LPS-induced expression of *Ccl2*. Analysis using luciferase reporter assay revealed that a Nrf2 binding site in the *Ccl2* 5' flanking region from -235 to +85 contributed to gene silencing of *Ccl2* by activation of Nrf2. Our findings suggest that the druggable Nrf2 pathway may be an effective target to combat obesity-associated inflammation in adipose tissue and its' concomitant metabolic disorders. Supported by AMED BINDS JP19am0101001 (MY), 19H05649 (MY), 16KK0195 (AU), NIH R35 CA197222 (TWK), JSPS OT 290125 (YY).