is itself a regulator of glucose homeostasis. Here, we determined whether the gut microbiome influences glucose homeostasis through effects on gut-derived serotonin. Using both pharmacological inhibition and genetic deletion of gut-derived serotonin synthesis, we find [1] that the improvements in host glucose handling caused by antibiotic-induced changes in microbiota composition are dependent on the synthesis of peripheral serotonin.

[1] The gut microbiome regulates host glucose homeostasis via peripheral serotonin. Proc Natl Acad Sci U S A. 2019 Oct 1;116(40):19802-19804. Martin AM, Yabut JM, Choo JM, Page AJ, Sun EW, Jessup CF, Wesselingh SL, Khan WI, Rogers GB, Steinberg GR, Keating DJ.

Neuroendocrinology and Pituitary

CASE REPORTS IN UNUSUAL PATHOLOGIES IN THE PITUITARY

A Case of Ectopic Neurohypophysis

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SUN-287

A CASE OF ECTOPIC NEUROHYPOPHYSIS

Pituitary stalk interruption syndrome (PSIS) is a congenital disorder of the pituitary gland. Symptoms at presentation may vary widely as this disease presents along a spectrum which includes; ectopic posterior pituitary, interrupted pituitary stalk or aplasia and hypoplasia of the pituitary gland. It is a heterogeneous disorder in terms of radiologic and clinical presentation. It can present clinically as an isolated pituitary hormone deficiency (most common being growth hormone deficiency) or as multihormonal deficiencies.

CASE PRESENTATION

Patient is a 34-year-old woman with history of primary amenorrhea who was evaluated by a gynecologist and was prescribed oral contraceptive pills which lead to her having a menstrual bleed for the first time in her life. She denied any difficulty with smell. She had undergone normal psychomotor milestones and highest level of education was high school. She had normal puberty with normal pubic and axillary hair growth, normal breast development but no menarche. Of note, patient has a short stature, height is 4 feet and 11 inches, and her biological parents are of no menarche. Of note, patient has a short stature, height is 4 feet and 11 inches, and her biological parents are of

Patient is currently being treated with hormonal replacement which is the main modality of treatment for ectopic neurohypophysis. She will need long term follow up as disease progression to pan-hypopituitarism is common.

CONCLUSION

PSIS is a rare syndrome with different phenotypic presentation depending on when the diagnosis is made; therefore, adequate follow up is indicated as the disease can progress from a single hormonal deficiency to pan-hypopituitarism.

Pediatric Endocrinology

PEDIATRIC SEXUAL DIFFERENTIATION, PUBERTY, AND BONE BIOLOGY

A Quantitative-PCR Based Rapid and Cost-Effective Diagnostic Method for Turner Syndrome and Its Variants

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SUN-084

Turner’s syndrome (TS) is a common aneuploidy diagnosed by peripheral blood karyotyping in patients. Karyotyping involves manual labour, time and costs. Quantitative real-time PCR (qPCR) is a rapid molecular diagnostic test for TS that yields results of up to 48 samples within two hours at low cost with reasonable accuracy. To assess the sensitivity and specificity of qPCR in the rapid diagnosis of TS, genomic DNA was isolated and estimated from peripheral blood from 50 TS patients(45,XO-23, XO/XX mosaics - 10, Isochromosome Xq-12, XO/XY mosaics - 5), 25 normal females(46,XX) and 5 normal males(46,XY). qPCR was done using 96 well plates, fast real-time PCR. 4 primers were used - two on Xp [SHOX and ARSE] and two on Xq (VAMP7 and KIST). Autosomal gene HBB was used as housekeeping gene. The ΔΔ CT method was used for calculation of the ‘X gene dose’ with respect to the housekeeping gene and X genes from normal females. Differences of doses of the four X-chromosomal primers in different karyotypes were analysed. ROC curves were plotted to determine cut-offs to discriminate the different karyotypes of TS from normal females. qPCR could distinguish classical TS from normal females with >95% sensitivity and specificity. SHOX gene primer was the best to diagnose TS of all karyotypes combined and also classical TS(XO) from normal females. qPCR could also identify non-classical TS with >92% sensitivity and specificity, the best primer being ARSE, for detecting both mosaics and isochromosomes. The cut-offs determined from our study corroborates with past similar studies.1,2 qPCR using an appropriate panel of primers on the short and long arms of X chromosomes can be a rapid and cheaper alternative to karyotyping to diagnose TS of different karyotypes. The choice of primers should be guided by the need for a more sensitive or specific test depending on the clinical scenario. If used as a neonatal screening test, SHOX should be the best primer. For diagnostic purposes, when the pre-test probability is low, a
more specific primer like SHOX would be more appropriate. However, when the pre-test probability is high, a sensitive primer for ruling out TS like VAMP7 is better. In case there is a high pre test probability of the patient having a non-classic TS rather than classic TS, ARSE should be used. This is the first study to show good sensitivity of qPCR in detecting non-classic TS of different karyotypes in addition to classic TS. References: 1. Ibarra-Ramirez M, Martinez-de-Villarreal LE. Clinical and genetic aspects of Turner's syndrome. Medicina universitaria. 2016 Jan 1;18(70):42-8.
2. Rocha MN et al. Applicability of real-time PCR methodology in the neonatal detection of Turner syndrome. Hormone and metabolic research. 2010 Aug;42(09):677-81.

Thyroid
THYROID DISORDERS CASE REPORTS II

Non-Adherence to Levothyroxine Treatment, a Condition Not to Be Forgotten, Should Be Assessed by Thyroxine Absorption Test
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SAT-505
Hypothyroidism due to non-compliance with levothyroxine (LT4) treatment is not infrequent (pseudomalabsorption). It should be considered in patients with persistent severe clinical and biochemical hypothyroidism even after excessive LT4 dose. The diagnosis can be confirmed by LT4 Absorption Test. We present 4 female patients (age range 21-44 years) with suspicion of (pseudo) malabsorption who underwent absorption test (3 patients with autoimmune hypothyroidism and one patient with hypothyroidism after total thyroidectomy due to Graves’ disease). They presented with persistent hypothyroidism (TSH 30 to >100 mU/L) even after gradual increase to excessive LT4 dose (400-700 μg daily). They denied non-compliance; drug and dietary interference with LT4 absorption and nephrotic syndrome were excluded. Two patients with autoimmune hypothyroidism underwent absorption test with their last daily LT4 dose as loading dose (700 and 200 μg) followed by hourly free T4 (fT4) determination for 6 hours, after an overnight fast. In one fT4 remained stable during the test (maximum fT4 increase +10% from baseline levels) indicating true malabsorption. New absorption test with combination of LT4 and ascorbic acid resulted in a fT4 raise +139% and further investigation revealed achlorhydria due to pernicious anaemia. The patient was treated with LT4 400μg x 2, Liothyronine 40μg x 2 and vitamin C in high doses. In the other patient, fT4 rose to maximum +71% from baseline 6 hours after the loading dose intake. She was diagnosed with pseudomalabsorption and became compliant and biochemically euthyroid with 150 μg/day LT4. Another two patients (1 with autoimmune, 1 with hypothyroidism after total thyroidectomy) underwent absorption test with a weight-adjusted weekly fasting LT4 dose (1,6 μg/kg of the body weight X 7) followed by hourly fT4 measurement for 5 hours. Peak fT4 reached a level of +290% and +309% of the baseline fT4 levels, respectively, 3 hours after administration of the dose. Both patients had pseudomalabsorption. They continued to deny non-compliance and were treated with once weekly supervised weight-adjusted LT4 over 6 consecutive weeks, resulting in TSH normalization. Pseudomalabsorption should be ruled out with LT4 absorption test in patients suspected of non-compliance with LT4 treatment, after drug/dietary interference, nephrotic syndrome and intestinal malabsorption are excluded. Different absorption protocols have been suggested with different loading doses (standard or weight-adjusted) and different duration (rapid 2-6 hours, long 5 weeks). An LT4 absorption peak with >70% increase in fT4 levels in 3 hours with a linear increase of fT4 in the first 1-1.5 hour is expected in the rapid test. In the long test normalization of TSH and fT4 is anticipated week 6 (1 week after the final dose). In case the patient remains non-compliant, treatment options include a single supervised weekly LT4 dose.

Neuroendocrinology and Pituitary
ADVANCES IN NEUROENDOCRINOLOGY

Effects of High-Fat Diet-Induced Obesity on Pulsatile LH Secretion and Hypothalamic KISS1 Expression in Female Rats
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SUN-239
Female obesity is associated with menstrual dysfunction leading to anovulation and infertility. It has recently been reported obesity-induced infertility is involved in the dysfunction of a kisspeptin neuron, a key player in reproduction via direct stimulation of gonadotropin releasing hormone (GnRH) and subsequent gonadotropin release in mammalian species. Previous studies reported that obesity due to high-fat diet (HFD) for 8 months induced a disruption in estrous cyclicity, caused by a decrease in Kiss1 (coding kisspeptin) expression in the hypothalamic arcuate nucleus (ARC) in female rodents. Here we showed the effects of shorter-term (4 months) HFD on pulsatile LH secretion and hypothalamic Kiss1 expression to show pathogenic mechanism underlying obesity-induced infertility. Female Wistar-Imamichi strain rats (7 weeks old) fed on either a standard diet (10% calories from fat) or a high-fat diet (45% calories from fat) for 4 months. Estrous cyclicity and body weight were monitored regularly. All animals were implanted with a jugular catheter and collected blood samples to analyze pulsatile LH secretion, after a week of the ovariecotmy with low-dose replacement estradiol to negate influence of changes in ovarian steroid levels and mimicking levels of plasma estrogen. On the next day of the blood sampling, rats were perfused with 0.05 M PBS followed by 4% paraformaldehyde and their brains were collected for in situ hybridization of Kiss1 and Gnrh1. The HFD-fed rats showed progressive increases in body weight, along with hyperphagia and adipose tissue accumulation, compared with control animals. Fifty-eight percent of the HFD-fed rats exhibited irregular estrous cycles, whereas remaining HFD-fed rats showed regular cycles. Two out of 7 rats showing HFD-induced irregular