Global Fatigue Score decreased from 5.6 (2.5) at BL to 3.5 (3.0) at W48, and to 3.8 (2.2) at W144 (both p<0.01). All 3 domains of the Brief Pain Inventory decreased with burosumab (W144 Pain Severity and Pain Interference p<0.05), indicating reduced pain. The SF-36 mean (SD) physical component summary score increased from 33 (10) at BL to 39 (10) at W48 (p<0.05) and to 41 (12) at W144 (p<0.01), indicating improved physical functioning. The mean (SD) number of sit-to-stand repetitions, an assessment of proximal muscle function, increased from 6.7 (4.2) at BL to 8.5 (4.2) at W48 (n=10; p<0.01). All subjects had ≥1 adverse event (AE). Two subjects discontinued: 1 to undergo chemotherapy to treat an AE of neoplasm progression and 1 failed to meet serum phosphorus dosing criteria and therefore received minimal burosumab dosing. There were 16 serious AEs in 7 subjects, all unrelated to drug. Of the 6 subjects with a serious AE of tumor progression/compression, 5 had a history of tumor progression prior to enrollment. There was 1 death, considered unrelated to treatment. In adults with TIO Syndrome, burosumab was associated with improvements in phosphate metabolism, osteomalacia, skeletal metabolism/fracture healing, physical functioning, fatigue, pain, and quality of life.

Cardiovascular Endocrinology

PATHOPHYSIOLOGY OF CARDIOMETABOLIC DISEASE

Elevation of Serum Kisspeptin in Hypertensive Women

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Kisspeptin and leptin have been shown to have an effect on the cardiovascular system. This study aimed to compare serum kisspeptin and leptin levels between the non-hypertensive (non-HT) and the hypertensive (HT) groups with or without body mass index matching, and determine correlations between systolic blood pressure or diastolic blood pressure with serum kisspeptin and leptin levels as well as clinical and adipocyte parameters. 30 female patients who underwent abdominal surgery were recruited. Blood samples, anthropometric data, and tissue samples of visceral and subcutaneous fat were obtained. Serum kisspeptin levels (ng/ml) (non-HT=1.01±0.1 vs. HT=1.53±0.19), body weight (kg) (non-HT=55.45±3.37 vs. HT=63.69±2.42), waist circumference (cm) (non-HT=78.01±2.49 vs. HT=84.89±2.40), hip circumference (cm) (non-HT=92.94±2.18 vs. HT=99.43±1.85), plasma glucose (mg/ml) (non-HT=55.45±3.37 vs. HT=63.69±2.42), plasma insulin (μM/ml) (non-HT=4.64±0.92 vs. HT=7.13±0.85), the homeostatic model assessment for insulin resistance (HOMA-IR) (non-HT=0.94±0.20 vs. HT=1.72±0.22), and height of visceral adipocytes (μm) (non-HT=72.64±6.75 vs. HT=90.25±4.52) were significantly higher but the quantitative insulin sensitivity check index (QUICKI) (non-HT=0.41±0.01 vs. HT=0.36±0.01) was significantly lower in hypertensive compared to non-hypertensive subjects (p<0.05 all). Systolic blood pressure had significantly positive correlations with diastolic blood pressure
(R=0.568), glucose (R=0.526), the HOMA-IR (R=0.387), and serum kisspeptin (R=0.569), but has a significantly negative correlation with the QUICKI (R=-0.414). Diastolic blood pressure had positive correlations with body weight (R=0.477), waist circumference (R=0.517), hip circumference (R=0.578), glucose (R=0.533), the HOMA-IR (R=0.415), and width (R=0.436) and height (R=0.439) of visceral adipocytes, but has a negative correlation with the QUICKI (R= -0.464). In conclusion, kisspeptin, obesity especially visceral adiposity, and insulin resistance might contribute to increased blood pressure in hypertensive subjects.

Thyroid

THYROID DISORDERS CASE REPORTS I

A Case of T3 Thyrotoxicosis

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Clinical vignette ENDOCRINE SOCIETY 2020
Title: A case of T3 thyrotoxicosis induced by a dietary supplement.
A 24 yo man consulted for a 2 weeks history of diaphoresis, fatigue, insomnia, palpitations and headache associated with a 20 pounds lost. The patient didn't have a goiter or any signs of orbitopathy.
The results revealed a free T3 level of 45.8 pmol/L upon arrival (normal (N) 3.4- 6.8 pmol/L), free T4 level of 6.4 pmol/L (N 11.0–22.0 pmol/L) and TSH level less than 0.005 mUI/L (N: 0.35 to 3.50 mUI/L). Facing those results, a complete review of the patient medication and natural product consumption was done. The patient revealed that he was using, since a month, a vegetable extracts nutritional supplement that didn't included iodine. He was asked to stop the nutritional supplement and propranolol 10 mg twice daily was prescribed. Thyroid function tests were done 3 days after. The results demonstrate a fT3 level of 4.6 pmol/L, a fT4 level of 5.6 pmol/L and a TSH that still suppressed. A thyroid scintigraphy was performed 7 days later and showed a homogeneous uptake of 18.5% (N 7.0% – 35.0%). We saw the patient 2 weeks later and we ordered another thyroid function test with TSH receptor antibodies, TPO antibodies and thyroglobulin. The results were the following: fT3 of 5.1 pmol/L, fT4 of 12.1 pmol/L, TSH of 2.31 mUI/L, thyroglobulin of 19.8 ug/L (N: 1.4 – 78) and normal levels of antibodies against TPO and TSH receptors. To confirm the contamination of the nutritional supplement by fT3 we used a plasma pool of normal patients in which we measured thyroid function tests at baseline and after we have added the nutritional supplement powder to reflect the dose suggested by the manufacturer. The results showed that fT3 level increased by 36.5%, fT4 by 11.2% and TSH didn't changed. The powder was then analyzed by an external laboratory that wasn't able to demonstrate the presence of fT3 nor fT4. The two diagnostic possibility facing those results were that the powder induced an interference