resistance and may have a cardioprotective effect, contrary to the traditional view that testosterone is detrimental to the heart. Our study aims to evaluate the effect of testosterone therapy on glycaemic control other cardiovascular risk factors, symptomatic benefit and quality of life in a randomised double-blind placebo controlled add-on of testosterone therapy to their normal hypoglycemic medication in hypogonadal men with uncontrolled type 2 diabetes. The study population includes 65 eligible men (140 screened) with poorly controlled diabetes (HbA1c between 53 and 80 mmol/mol) and confirmed hypogonadism by early morning [0800−1200h] total testosterone [TT] ≤12 nmol/L or calculated free testosterone ≤255 pmol/L on two occasions ≥1 week apart, with at least two symptoms of hypogonadism. The trial is divided into 2 phases. Phase 1: patients are randomly assigned to either treatment (depot testosterone undecanoate) arm or the placebo arm for 6 months. Phase 2: open label phase for 6 months with subjects on placebo on placebo initiated on to testosterone therapy while subjects in the treatment group continue to receive treatment for the 12 month duration. No change to anti- glycaemic therapy was made during the first phase of the study. The primary endpoint is HbA1c. Secondary endpoints include body composition (bioelectrical impedence DEXA scan), HOMA-IR, lipid profile, blood pressure (24 hr BP monitor), carotid media intima thickness, monocyte mRNA cytokine expression, Questionnaires include AMS (Aging Male Symptom Score), IIEF-5(International Index of Erectile Dysfunction), SF36-Quality of life, Mini mental score, New questionnaire for hypogonadism in diabetes (to be validated), NERI (New England Research Institute) hypogonadal screener. Baseline data indicate the mean age 59 (42-77) years. Mean Duration of diabetes was 8.6(0-21) years. 18 men were on Insulin. The remaining 47 men were either diet controlled or on oral hypoglycaemic medications. 9 men had pre-existing history of MI and 4 had history of angina. Mean HbA1c at baseline was 65(53-80) mmol/mol. Mean total testosterone level was 8.9(2.1-16.9) nmol/l. Mean weight and BMI at baseline were 107(71-187) kg and 34.5(24-52) respectively. Mean waist circumference was 115.7(46-160) cm The primary aim of this is to determine if testosterone therapy improves glycemic control in men with uncontrolled diabetes. Secondly to assess beneficial effects on specific cardiovascular parameters as well as QOL. This could have a major clinical implication on how we treat patients with hypogonadism and type 2 diabetes.

Adrenal

ADRENAL CASE REPORTS I

The Use of ^{11}C-metomidate PET-CT to Detect Unilateral Primary Hyperaldosteronism

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SAT-196

Background

Identifying causative adrenal lesions presents a significant diagnostic burden for physicians and radiologists. We describe the use of radiolabelled metomidate to lateralise primary hyperaldosteronism.

Case presentation

A 52-year old Chinese man with a 5-year history of hypertension was referred for hypokalemia [K 2.7 mmol/L (3.6 - 5.0)]. He had been on Telmisartan 80 mg and Amlodipine 10 mg daily and blood pressure at home ranged 110-120 / 70-80 mmHg. There was no history of poor oral intake, persistent diarrhea or vomiting, and he was not on any other prescription or alternative medications. There was no significant family history of hypertension or sudden cardiac death. Clinic blood pressure was 140/84 mmHg. There were no features suggestive of Cushing's syndrome.

Repeat biochemical tests confirmed hypokalemia (K 3.1 mmol/L), and associated raised bicarbonate 37.3 mmol/L [19 - 29]. Magnesium and creatinine were normal. Aldosterone-renin Ratio was elevated at 8.1 (serum Aldosterone 611 pmol/L [97.3 - 834.0], active renin 2.7 pg/ml [1.8 – 59.4]). Post-saline infusion, non-suppressible serum aldosterone levels of 1137 pmol/L was demonstrated, consistent with autonomous aldosterone production.

A computed tomography of the adrenal revealed a 2.3 cm x 1.9 cm nodule on the left adrenal gland consistent with lipid rich adenoma.

Adrenal vein sampling (AVS) under continuous synacthen infusion was performed. Adrenal to peripheral cortisol ratio was ≥10 for either adrenal veins, confirming cannulation of the adrenal veins. Aldosterone-cortisol ratios showed lateralization to the left adrenal gland (lateralization ratio of 10.35). There was contralateral suppression of the right adrenal gland with ratio of 0.41.

^{11}C-Metomidate PET-CT scan demonstrated a maximum standardised uptake value (SUV_{max}) of 26.8 over the left adrenal nodule, while the SUV_{max} of the right adrenal gland was 16.2. Ratio of the left to right adrenal gland SUV_{max} was 1.65 (above the threshold of 1.25); and was concordant with AVS. This confirmed that the patient had a left functional adrenal adenoma responsible for hyperaldosteronism.

Our patient underwent a left adrenalectomy, and histology was consistent with adrenal cortical adenoma. Prior to surgery he required 72 mmol/l of potassium supplementation daily to maintain K levels of 3.3 – 4.0 mmol/L. Two weeks post-operatively, he was normokalemic (K 4.9 mmol/L) without potassium supplementation. Serum aldosterone normalized to 159.3 pmol/L (active renin 9.3 pg/ml). Blood pressure is well controlled on amlodipine 5mg daily.

Conclusion

Targeted molecular imaging such as ^{11}C-Metomidate PET-CT could aid localisation of functional adrenal disease to guide definitive surgical management. In the future, this could obviate the need for invasive and technically complex procedures like AVS.

Diabetes Mellitus and Glucose Metabolism

CLINICAL AND TRANSLATIONAL GLUCOSE METABOLISM AND DIABETES

Pattern of C-Peptide Response to Oral Glucose Tolerance Test: Interest and Cut-Off Values

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MON-623

Introduction: Oral glucose tolerance test (OGTT) allows classification of subjects in 3 groups, depending on glycaemia 120 minutes after 75g glucose ingestion: normal (glycaemia < 1.4 g/L), glucose intolerant (1.4-2 g/L) and diabetic (>2g/L). Five insulin profiles following OGTT associated with different incidence rates of diabetes over 10 years of follow-up have also previously been described (Kraft J et al, *Laboratory Medicine,* 1975; Hayashi T et al, Diabetes Care.2013). Insulin measurement is very sensible to hemolysis and can advantageously be replaced by C-peptide determination. However, little is known about C-peptide reference values and response to OGTT.**Material and Methods:** 128 patients were included to evaluate glyceamia (COBASe801® ROCHE Diagnostics, France), insulin and C-peptide (LiaisonXL®, Diasorin, France) responses to OGTT.**Results:** According to Hayashi classification, 23 (18%) patients of the whole cohort harbored a physiological insulin response corresponding to profile I (peak of insulin during OGTT at 30 min and higher insulin level at 60 vs. 120 min). Others presented 5 pathological profiles: 14 (11%) patients were classified in profile II (peak of insulin at 30 min and lower or equal insulin level at 60 vs. 120 min), 56 (44%) in profile III (peak of insulin at 60 min), 26 (20%) in profile IV (peak of insulin at 120 min and lower insulin level at 30 vs. 60 min), and finally 9 (7%) in profile V (peak of insulin at 120 min and higher or equal insulin level at 30 vs. 60 min). Only 4 different mean C-peptide profiles emerged from the subgroups previously defined by insulin profile, mean C-peptide profile being substantially similar to mean insulin profile. The only major difference relied on a similar C-peptide profile corresponding to a growing curve from T0 to T120 in both patients with insulin profile IV and V. Mean and 95% confidence interval of C-peptide value at the different times of OGTT were also calculated in the subgroup of patients with both normal glycemic and insulin (pattern I) responses to propose reference values: respectively T0: 0.53 (0.26-0.77); T30: 2.2 (1.24-3.29); T60: 2.26 (1.36-3.68); T120: 1.88 (0.84-2.62) nmol/L. **Conclusion:** C-peptide response to OGTT profile seems to give globally the same information as insulin profile and should therefore also be predictive of the risk type 2 diabetes in case of hemolyzed samples. The slight differences observed between insulin and C-peptide profiles can be explained by their different metabolic pathways, insulin being quickly degraded in the liver and C-peptide undergoing a longer renal elimination. This work also allows us to propose for the first-time reference values for C-peptide at the different times of OGTT using Liaison XL®.

Pediatric Endocrinology

PEDIATRIC PUBERTY, TRANSGENDER HEALTH, AND GENERAL ENDOCRINE

Do Low Sex Hormone Binding Globulin Levels in Newborns Predict Weight Gain in Infancy and Early Childhood?

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SUN-077

Background: SHBG levels are low in obesity, and low SHBG levels are a biomarker for the development of T2DM and the metabolic syndrome. We sought to determine whether low SHBG in newborns will predict childhood obesity. **Methods:** We studied 94 healthy, singleton, full-term newborns, and measured their length, weight (BW), waist circumference, and skinfold thicknesses. We collected cord blood as well as day 2 venous blood samples for the measurement of SHBG and insulin (ALPCO, Salem NH). Maternal age, pre-pregnancy weight, pregnancy weight gain, and glucose screening test results were obtained from obstetrical records. Mothers with chronic diseases were excluded from the study. When babies were 2 years old, we administered a questionnaire to collect information about their eating, sleeping, screen viewing habits, and anthropometric measurements at ages 6, 12, and 24 months (n=47). Overweight was defined as a BMI SDS of ≥1 and <2.0, and obesity as ≥2 SDS. We used the Shapiro-Wilk test to determine if variables were normally distributed. Data were analyzed using the Mann Whitney U and Wilcoxon signed-rank tests, and by Pearson or Spearman correlation analyses. We report non-normally distributed variables as medians and interquartile ranges (IQR). Because of skewed distributions, log 10 transformed values for SHBG were used in the regression analyses. **Results:** SHBG levels on day 2 were significantly higher than in cord blood [22.0(28.7-16.9) vs. 19.0(24.6-14.5) nmol/L, p<0.001], whereas insulin levels were higher in cord blood than in day 2 samples [3.2(5.3-2.0) vs. 1.5(2.2-0.8) μIU/mL, p<0.001]. SHBG and insulin levels were similar in male (n=44) and female (n=50) babies at all time points. Babies with Ponderal index values in the highest quartile had lower day 2 SHBG [18.2(22.1-16.7) vs. 24.3(30.3-18.2) nmol/L, p=0.02] and higher cord blood insulin levels [5.0(7.4-2.6) vs. 2.9(4.8-1.5) μIU/mL, p=0.04] than the remainder of the cohort. At age 2 years, 32% (15/47) of babies were overweight or obese, 60% (28/47) were breastfeeding, 58% (27/47) were watching TV or iPads, and 55% (26/47) were eating sweet snacks. Toddlers watching TV or iPads (p=0.008), or eating sweet snacks (p=0.04) were heavier than their peers. Neither cord blood nor day 2 SHBG or insulin levels correlated significantly with any of the anthropometric measurements in the newborns. On the other hand, day 2 SHBG levels correlated positively with weight at 6 (r=0.311, p=0.04) and 24 months (r=0.353, p=0.02) of age. These associations remained significant after adjusting for gender, BW, gestational age, breastfeeding status and fruit juice intake at 6 months (R^2=0.28, p=0.048) and for gender, BW, gestational age, breastfeeding status, sweet snack intake and screen viewing habits at 24 months (R^2=0.33, p=0.046). **Conclusion:** Although the heaviest babies had lower SHBG levels at birth, low SHBG did not predict overweight at age 2 years.

Adrenal

ADRENAL - CORTISOL EXCESS AND DEFICIENCIES

Structural Instability as an Underlying Pathomechanism in Congenital Adrenal Hyperplasia

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