resonance spectroscopy for hepatic (HCL) and myocardial (MYCL) lipid content analysis. The control group (CON) is conducted by age, sex and BMI matched healthy individuals. **Results:** Until now we included 26 MtF,14 FtM patients and 12 age and BMI matched healthy controls. The mean age was comparable in all 3 groups (MtF 30.12±2.31, FtM 29.72±1,91, CON 30,23±1.22 as well as BMI (22.59±3.81, 21.62±2.53, 21.33±1.20 kg/m2, p=ns, respectively). The mean hormone therapy duration was similar in both groups (MtF 4.58±1.20 vs FtM 2.35±0,95, p=0,29). HOMA Index did not significantly differ between the groups (MtF 1,78±0,92 vs FtM 1,96±1.22 vs CON 1,8±1.01, p=0,3 vs 0,4 vs 0,3 respectively). HCL was significantly higher in MtF than FtM (1,50±0,41 % vs 0.54±0,33 %, p=0,022, respectively). We also found a significant correlation between ejection fraction (EF) and Testosterone levels (Spearmans Rho 0,80, p=0.002). **Conclusio:** These preliminary data could indicate a positive effect of Testosterone therapy on heart function. Contradictory to current data we found a higher HCL in MtF than FtM suggesting a not so protective estrogen effect when looking at the liver. Long-term studies are warranted to assess whether cross-sex HT results in different outcomes regarding cardiovascular disease.

Diabetes Mellitus and Glucose Metabolism

CLINICAL AND TRANSLATIONAL STUDIES IN DIABETES

Course of Puberty and Growth Spurt in Boys with Type 1 Diabetes

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MON-659

Course of puberty and growth spurt in boys with type 1 diabetes

Background: Data on the course of puberty and pubertal growth in boys with Type 1 diabetes (T1D) are sparse.

Objectives: To study the course of puberty, pubertal growth and final height in boys with T1D as well as possible factors affecting these.

Methods: In this retrospective longitudinal study, 68 boys diagnosed with T1D between 1996-2009 who were prepubertal at diagnosis and had completed puberty served as the cohort. Collected were data on anthropometric measurements, Tanner stage, and HbA1c levels from diagnosis to final height (F-Ht). F-Ht was compared to parental height and to the data of the national health survey

Results – In the study cohort final height-SDS was lower than that at diagnosis. It was similar to parental Ht-SDS as well as to that of the national health survey (p=0.126). F-Ht was inversely related to average HbA1c during puberty (R=-0.27, p=0.045). Boys who presented with diabetic ketoacidosis at diagnosis were shorter than those who did not throughout the entire follow-up. Age at onset of puberty was significantly related to the age of maternal menarche (R=0.44, p=0.01) and to HbA1c levels in the year preceding puberty onset (R=0.36, p=0.01). Total pubertal growth was inversely related to HbA1c levels in the year preceding onset of puberty (average R=-0.3, p=0.03)

Conclusions: Boys with T1D diagnosed before puberty achieve final height similar to that of their parents and that of the general population. Diabetic ketoacidosis at the diagnosis is associated with diminished F-Ht. Age of pubertal onset and F-Ht are affected by genetic factors as well as by glycemic control before and during puberty.

These results emphasize the importance of tight metabolic control in adolescents, to enable growth within the genetic target.

Pediatric Endocrinology

PEDIATRIC ENDOCRINE CASE REPORTS II

Ketotic Hypercalcemia; A Possible Side Effect of Managing Refractory Epilepsy with Ketogenic Diet

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MON-074

Background: Patients with epilepsy have multiple risk factors for low bone mass, including immobility, reduced muscle mass, and use of multiple anticonvulsant medications. The ketogenic diet (KD) has been used as an effective treatment of refractory epilepsy for decades, with multiple reports in medical literature describing adverse effects on bone and mineral metabolism including gradual loss of bone mineral density. There has been only one report of hypercalcemia in three children on the KD for refractory seizures. We now describe another case, highlighting the importance of considering the KD as a cause of hypercalcemia.. **Case presentation:** A 14-year-old girl with Rett syndrome, epilepsy, global developmental delay and gastrostomy tube dependency presented to the emergency room with marked dehydration. She had been on the KD for several years due to refractory epilepsy. Her parents had recently noticed thick oral secretions. The only change in her management plan was the recent change of her formula from Ketocal 3:1 to Ketocal 2.5:1. Lab studies showed hypercalcemia 15 mg/dL (ref 9.2-10.7) with ionized calcium of 7.99 mg/dL (ref, 4.8-5.2). She had normal serum calcium levels on multiple previous occasions, including 10.2 mg/dL 4 months prior to presentation. Other studies included increased BUN 37 mg/dL (ref, 7-21) and Creatinine 2.31 mg/dL (ref, 0.53-0.8). She had a low normal PTH 14 pg/ml (ref, 8-72), PTH-related peptide 0.6 pmol/l (ref, < 4.2), and 25-hydroxyvitamin D 66 ng/ml (30-100). 1,25-dihydroxyvitamin D level was low at 12.5 pg/ml (ref, 19.9-79.3), with increased urine calcium/creatinine ratio 1.00 mg/mg creat (Ref, 0.02-0.26). Beta Hydroxybutyrate was 3.45 mmol/l (ref, 0.02 - 0.27), without major change over the last year. Renal US was normal. She received IV hydration with improvement in serum calcium, BUN, and creatinine and was discharged on increased intake of free water and adjustment of KD to maintain Beta Hydroxybutyrate around 2 mmol/l. Her serum calcium currently ranges 10.5- 12 mg/dl, and will soon begin therapy with subcutaneous calcitonin. **Conclusion:** The KD has adverse effects on bone and mineral metabolism, and can lead to severe