14.48 x 98.40 ± 27.48 mg/dL (p=0.359), HbA1c 5.83 ± 0.33 x 6.21 ± 1.18% (p=0.185), HOMA-IR 3.61 ± 1.28 x 5.31 ± 4.74 (p=0.160), TC 170.87 ± 39.36 x 180.82 ± 34.51 mg/dL, LDL 94.67 ± 26.63 x 105.60 ± 30.85 mg/dL, HDL 53.37 ± 18.50 x 50.84 ± 10.32 mg/dL, triglycerides 114.12 ± 38.84 x 127.30 ± 75.04 mg/dL, and hs-CRP 8.51 ± 6.50 x 7.51 ±6.52 mg/L (p=0.792). CONCLUSION: Women with SARC and recommendation for BS when compared to women without SARC had similar anthropometric, metabolic and BFP parameters.

Adrenal
ADRENALECOTOMY FOR ALDOSTERONOMA

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SUN-196
Introduction
Following unilateral adrenalectomy, some patients experience persistent hyperkalemia. This has been attributed to hypoaldosteronism due to severe suppression of aldosterone synthesis in the contralateral adrenal gland. Additionally, excess aldosterone leads to glomerular hyperfiltration masking kidney dysfunction, which can then manifest after cure with CKD-related hyperkalemia.

Case Presentation
We report a case of 51-year-old male who was diagnosed with hypertension in his late 30s. He required a beta-blocker and calcium channel blocker (CCB) for 10 years, and eventually developed hypertensive nephropathy. With worsening lower extremity edema, he was switched from a CCB to an angiotensin receptor blocker. Soon afterwards, he presented with hypertensive emergency and was discovered to have significant hypokalemia (K 2.1 mmol/L), prompting work up for primary aldosteronism.

Biochemical evaluation revealed an elevated aldosterone to renin ratio of 38 [(ng/dL)/(ng/mL/hr)] and adrenal protocol CT scan revealed a 1.9 cm left adrenal nodule with benign characteristics. Adrenal vein sampling showed marked lateralization of excess aldosterone to the left adrenal gland, with proper catheter placement demonstrated in each adrenal veins (5-fold cortisol gradient bilaterally).

He was started on spironolactone and 6 weeks later, underwent a successful laparoscopic left adrenalectomy. Spironolactone was discontinued. Serum K level was normal at 4.8 mmol/L immediately postoperatively. Ten hours later, his K went up to 6.6 mmol/L which was confirmed by repeat blood work, accompanied by worsened renal function (Cr 2.5 mg/dL up from a of baseline 2.0). His hyperkalemia persisted in the 5.0 – 6.0 range postoperative hyperkalemia in this patient to a combination of hypoaldosteronism due to deep suppression of the mineralocorticoid production of the contralateral adrenal, as well as unmasking of more severe kidney dysfunction than was he previously thought to have once aldosterone excess was withdrawn.

Thyroid
GRAY'S DISORDERS CASE REPORTS I

Graves’ Disease Induced Renal Tubular Acidosis
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SUN-512
Thyroid gland can affect kidney function in different ways. Thyroxine as a master hormone of metabolism and growth works in many cellular levels include the renal tubules. We present a 34-year-old Emirati gentleman who presented with multiple episodes of hypokalemic periodic paralysis. Blood test revealed thyrotoxic state, with highly positive serology for thyroid peroxidase, anti-thyroglobulin and thyrotropin receptor antibodies. Thyroid uptake scan confirmed homogenous diffuse uptake consistent with toxic diffuse goitre [Graves’ disease]. In view of recent fracture, bone profile and DXA scan were done. Investigations revealed vitamin D deficiency and below expected for age bone mass density.

The patient was started on symptomatic treatment with propranolol, IV and oral potassium along with IVF hydration. Routine blood work during admission showed a persistent normal anion Gap metabolic acidosis, serum bicarb 15 mmol/L. 24 hours urine electrolytes revealed normal potassium, sodium, high magnesium, low calcium and PH levels. Biochemical lab results suggested type 1 renal tubular acidosis. As the patient had hypokalaemia, high urine magnesium and low urine calcium and limbs weakness, Gitelman Syndrome was considered in the differential diagnosis. Whole Exome Sequencing (CentoXome GOLD®) was sent which came back negative. The following gene panels were studied: Renal tubular acidosis panel: ATP6V0A4, ATP6V1B1, CA2, EHHADH, HNF4A, SLC34A1, SLC4A1, SLC4A4. Bartter Syndrome panel: ATP6V1B1, BNSD, CA2, CASR, CLCNKA, CLCNKB, CLDN16, CLDN19, FXYD2, HSD11B2, KCNJ1, KCNJ10, KLHL3, NR3C2, SCN1A, SCN1B, SCNIN, SLC12A1, SLC12A2, SLC12A3, SLC4A1, SLC4A4, WNK1. Gene related to Gitelman syndrome: SLC12A3

Renal tubular acidosis was treated with KCL 600mg PO TID, Na bicarb 1200mg PO BID and spironolactone 25 mg PO OD. The patient received radioactive iodine (RAI) as
Diabetes Mellitus and Glucose Metabolism

METABOLIC INTERACTIONS IN DIABETES

Bypassing Skeletal Muscle Lipid Handling Deficiencies as a Therapy for Metabolic Disease

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SUN-653

Metabolic diseases and their serious sequelae such as non-alcoholic fatty liver disease (NAFLD) pose a substantial clinical burden. It is now well recognized that skeletal muscle is a major site for the metabolism of all major macronutrients, and derangements in these muscle processes significantly contribute to metabolic disease. Studies over the last 15 years have identified the transcription factor Krüppel-like factor 15 (KLF15) as an important regulator and effector of metabolic processes across various tissues, and furthermore, genome-wide studies have identified human KLF15 variants with increased body mass index and diabetes. Given the importance of skeletal muscle in maintaining metabolic homeostasis, we generated a skeletal muscle specific KLF15 knockout (K15-SKO) mouse to study the role of skeletal muscle KLF15 in regulating systemic metabolism. We found that this animal is prone to developing obesity and insulin resistance at baseline, a phenotype that is greatly exacerbated in response to high fat diet (HFD). Strikingly, K15-SKO mice show a propensity toward developing NAFLD, as demonstrated by increased micro- and macrovesicular steatosis, hepatocellular ballooning, increased hepatic fatty acid and triglyceride deposition, and elevated Cd36 expression. A potential cause of NAFLD is the accumulation of excess lipids and lipid intermediates due to defects in the lipid flux pathway in extrahepatic tissues. Indeed, we see defects in the expression of genes involved in the carnitine shuttle and a paucity of long-chain acylcarnitines in K15-SKO skeletal muscle. Furthermore, RNA sequencing of skeletal muscle from K15-SKO mice shows downregulation in a number of pathways involved in lipid handling. This indicates that KLF15 serves as a novel extrahepatic molecular regulator of hepatic health. It has been previously shown that a diet rich in short-chain fatty acids (SCFA) can bypass defects in lipid handling and ultimately improve metabolic health. To explore this therapeutic avenue, we gave K15-SKO mice either normal chow (NC) or a SCFA-rich diet for 7 weeks. We observed decreased weight gain and improved glucose homeostasis in SCFA-rich diet fed mice. In addition to being a preventative strategy, SCFA-rich diets may also serve as a potential therapy to rescue from metabolic disease. To this end, we gave K15-SKO mice HFD for 5 weeks followed by 7 weeks of either NC or SCFA-rich diet. We observed that providing SCFAs can improve metabolic health and ameliorate the phenotype seen due to defects in skeletal muscle lipid handling: mice given SCFA-rich diet following HFD had significantly decreased weight gain and improved insulin sensitivity. These studies demonstrate that skeletal muscle KLF15 serves as an important regulator of lipid flux and hepatic health, and that SCFA-rich diets are a promising candidate for metabolic disease resultant of impaired lipid handling.

Diabetes Mellitus and Glucose Metabolism

DIABETES TECHNOLOGY

Self-Reported Psychological Stress and Glucose Variability in Type 1 Diabetes on Sensor Augmented Pump over 5 Weeks

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SAT-641

Self-reported psychological stress and glucose variability in Type 1 Diabetes on sensor augmented pump over 5 weeks

Introduction: Patients and their families and medical providers have assumed that psychologic stress impacts glucose control in T1D (Type 1 Diabetes) though studies providing confirmatory evidence in real world settings are, to our knowledge, lacking. We hypothesized that self-reported psychologic stress worsens glucose control in T1D.

Method: We studied 20 adults with T1D on continuous glucose monitor (CGM), sensor augmented insulin pump (SAP) prospectively at 2 clinical research centers. Patients reported psychological stress through stress diaries for 5 weeks on a severity scale of 1-7 using hard copy logs including time of onset and offset of stress and severity. For analytic purpose, grades 1-4 are classified as mild and grades 5-7 as severe.

Results: Baseline characteristics were age 44.9±15.0 years, F/M 12/8, HbA1c 6.8 ± 0.7%, and diabetes duration of 22.9±15.9 years. We analyzed glucose variability during days of stress versus days without stress. During a 24 hour period, patients experienced less hypoglycemia during days with stress versus days without stress (p value 0.03). During the 5 week period, patients reported 23 ± 19.5 events. We analyzed the