the ultimate treatment for Graves' disease. He developed hypothyroidism post RAI ablation and commenced on levothyroxine. Improvement of the metabolic acidosis was noticed in line with improvement of thyroid function. Na bicarb and spironolactone tablets were stopped eventually as the patient was euthyroid clinically and biochemically.
Overt hyperthyroidism is associated with accelerated bone remodelling, leading to hypercalciuria, which can predispose to nephrocalcinosis and renal tubular damage, and therefore causes type 1 renal tubular acidosis. Once the patient becomes euthyroid, bone remodelling and urine calcium return to normal levels and that would correct the renal acidosis.
This case report serves to highlight the effect of Graves' disease on renal tubules which may result in type 1 renal tubular acidosis. This effect could be reversible with normalization of thyroid function.

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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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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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