arcuate nucleus of the hypothalamus. This gene targeting strategy leaves pituitary *Pomc* expression unaffected. These mice are hyperphagic starting at weaning, and develop progressive obesity, infertility and insulin resistance over their lifetime. RM-493 (setmelanotide) is a melanocortin-4 receptor agonist that has shown promise in treating humans with *Pomc* null mutations. In this preclinical study, we investigated the effects of chronic RM-493 treatment using subcutaneously implanted osmotic minipumps in two groups of male mice: Arc-*Pomc* knockout mice, fed regular chow throughout the study period, and their wildtype counterparts, fed a 45% high-fat diet. Each of these groups of mice was randomized into three treatment cohorts at weaning: one that was given RM-493 throughout the entire study period (4–24 weeks of age, "RM-493" group), one that was given RM-493 only for the first 4 weeks of the study (4–8 weeks of age, "switch" group) and then switched to vehicle, and one cohort that received vehicle for the entire study ("vehicle" group). We serially measured body weight, food intake, body composition, glucose tolerance, insulin tolerance, and several measures of metabolism using the Comprehensive Lab Animal Monitoring System, including oxygen consumption, energy expenditure, ambulatory activity and lipid and glucose oxidation.

Among other results, at the end of the study (24 weeks of age), Arc-*Pomc* knockout mice in the RM-493 group weighed significantly less than either the switch or vehicle groups ($p<0.05$). Arc-*Pomc* knockout mice on RM-493 also had higher energy expenditure when compared to the switch and vehicle groups ($p<0.05$). In addition, RM-493 improved the glucose-insulin index for Arc-*Pomc* knockout mice ($p<0.05$). According to our preliminary results, wildtype mice on high-fat diet, treated chronically with RM-493, did not differ in any of these measurements from their switch and vehicle groups.

We conclude that the obesity syndrome caused by a loss of hypothalamic *Pomc* expression was completely blocked by RM-493 treatment started before the onset of obesity, with no apparent desensitization to the drug's action over 20 weeks. However, the beneficial effects of a single month's treatment were steadily reversed within one month after switching to vehicle treatment. In contrast to the dramatic effects of RM-493 in the genetic obesity syndrome, at this time, there does not appear to be any phenotypic changes in wild-type mice with RM-493 administration on the development of obesity or secondary metabolic disruptions in response to high-fat diet consumption.

Diabetes Mellitus and Glucose Metabolism

DIABETES COMPLICATIONS II

Diabetic Amyotrophy; A Rare Cause of Muscle Weakness

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Background: Diabetic amyotrophy is a rare complication of type 2 diabetes mellitus. There is little existing evidence contributing to projected outcomes for patients recovering from diabetic amyotrophy.Clinical Case: A 42 year-old man presented with lower extremity muscle pain and progressive proximal muscle weakness over 8 months. He developed asymmetrical muscle weakness in the lower extremities with diffuse pain and sensitivity to touch. He also had 80 pounds weight loss, diarrhea, and erectile dysfunction over the same time period. He had a past medical history of asthma, chronic migraines, and type II diabetes mellitus with A1c 7.1. His medications included high dose prednisone to treat his chronic migraines and asthma. Exam revealed generalized muscle atrophy, asymmetric proximal weakness, areflexia, with sensory loss in bilateral lower limbs.ESR, CRP, ANA, anti-HMG CoA reductase, CK, aldolase, SPEP, and myomarker panel were all within normal limits. Treponema pallidum and Bartonella serologies were negative. CSF evaluation was not suggestive of any demyelinating or neuromuscular disease. Full body STIR MRI demonstrated muscle edema in abductor, gluteus minimus, and paraspinal muscles bilaterally. EMG testing revealed acute to subacute active asymmetrical polyradiculoneuropathy and evidence of chronic proximal myopathy.Based on clinical presentation, EMG findings, and lack of evidence to support alternative diagnoses, he was diagnosed with diabetic amyotrophy and was started on IVIG and methylprednisolone with improvement in pain but very minimal improvement in weakness. Unfortunately, the expected clinical course following a diagnosis of diabetic amyotrophy is one of minimal improvement with treatment, as was the case in our patient.Conclusion: Diabetic amyotrophy is a rare complication of type 2 diabetes mellitus which typically presents with muscle weakness followed by severe pain in the thighs, hips, and buttocks. Compared with other neurologic complications of diabetes, amyotrophy is relatively uncommon, affecting approximately 1 percent of patients. This low prevalence and the broad differential for proximal muscle weakness makes it challenging to diagnose. It remains a diagnosis of exclusion, though EMG studies showing polyradiculoneuropathy in the proximal leg musculature is suggestive. Clinical improvement is slow and often incomplete. Physical and occupational therapy are a mainstay of treatment which may also include IVIG and steroids aimed at treating associated pain. Endocrinologists should have a high clinical suspicion for diabetic amyotrophy in the appropriate clinical context. When considering the diagnosis and discussing treatment options with patients, this case highlights the important role of endocrinologists discussing expectations associated with projected outcomes while attempting to manage diabetic amyotrophy.

Neuroendocrinology and Pituitary

CASE REPORTS IN UNUSUAL PATHOLOGIES IN THE PITUITARY

Lymphocytic Hypophysitis Mimicking Tolosa Hunt Syndrome

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Introduction:

Lymphocytic hypophysitis often presents with headache, hypopituitarism and visual disturbance, the latter from