Further long-term studies to establish the safety and efficacy of PSCK9 inhibitors post-transplant are needed.

Bone and Mineral Metabolism

BONE AND MINERAL CASE REPORTS II

Hypercalcemia During Teriparatide Therapy

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Background: Teriparatide (TPTD) is a recombinant PTH analog used as an anabolic agent in the treatment of osteoporosis that stimulates bone formation and activates bone remodeling. The most common side effects are nausea, vomiting, hypertension and dizziness. Transient hypercalcemia is a known adverse effect which is usually seen a few hours after administration and resolves within 16 hours. However, marked late hypercalcemia is a rare event and may be of concern in clinical practice.

Clinical Cases: **Case 1** is a 54-year-old man with a history of osteoporosis (lumbar spine T-score of -2.8), previously treated with bisphosphonates and who had been on a course of TPTD for about 6 months in but had not been consistent in taking the medication. Prior to subsequently restarting TPTD, his initial labs were notable for a normal Ca 9.3 mg/dl (8.5 - 10.1 mg/dl), vitamin D 25 OH 49 ng/ml (30.0 - 100.0 ng/ml) and PTH 41.3 pg/ml (8.7 - 77.1 pg/ml). Six months into the treatment, he was noted to have asymptomatic hypercalcemia of 11.2 mg/dl approximately 24 hrs after the last TPTD injection. A repeat calcium of 10.7 mg/dl was obtained while still on therapy with TPTD with normal levels of vitamin D 25 OH of 45 ng/ml.

Case 2 is a 75-year-old woman with a history of osteopenia and severe scoliosis, who had been on a course of raloxifene and then preventive doses of alendronate previously. Prior to starting TPTD, her Ca levels were normal at 9.3 mg/dl, PTH was 24 pg/ml and vitamin D 25 OH was 33 ng/ml. However, six months into the treatment she was noted to have elevated Ca of 12.5 mg/dl (24 hrs after the last TPTD dose), with low levels of vitamin D 25 OH of 24.2 ng/ml. Ca levels returned to the baseline of 9.3 mg/dl when TPTD was held.

Conclusion: Teriparatide has a long track record of safety and does have the rare side effect of hypercalcemia. 1-3% of patients may have mild hypercalcemia after administration. Intake of calcium and vitamin D should be monitored at the start of therapy given these concerns. Although almost never a cause of discontinuation of treatment in clinical practice, it is important to be aware of this side effect in patients who may be at risk of complications of hypercalcemia.

Diabetes Mellitus and Glucose Metabolism

CLINICAL STUDIES IN OBESITY, DIABETES RISK, AND CARDIOVASCULAR OUTCOMES

Potential Utility of the Mixed Meal Test for Differential Diagnosis of Partial Lipodystrophy from Common Type 2 Diabetes and Truncal Obesity

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Background: Better clinical tools are needed to improve the differential diagnosis of partial lipodystrophy (PL) from type 2 diabetes (DM) with truncal obesity. Here, we investigated differences in metabolic parameters during a mixed meal test in PL and DM patients to determine if this test may have a role in this regard. **Methods:** We retrospectively evaluated data collected from 17 PL patients (4M/13F, ages 12-64) and 20 DM patients (13F/7M, ages 24-72) with truncal obesity, who also had nonalcoholic fatty liver disease. All patients underwent a Mixed Meal Test (MMT) with 474 ml of Optifast (320 kcal, 50% carbs, 15% fat, and 35% protein). Blood was collected before and at 30, 60, 90, 120, and 180 minutes post-meal to measure glucose, insulin, free fatty acids (FFA), triglycerides, inflammatory markers, GIP, GLP-1, PYY, and Ghrelin. All samples of the same cohort were run at the same time in duplicates and results were averaged. Mixed linear models were constructed to compare PL and DM cohorts taking into account within-subject effects. Data were controlled for BMI, sex and age, and glucose when necessary. **Results:** Patients with PL had higher glucose and triglyceride levels throughout the MMT at all-time points ($p < 0.05$). While the glucose levels showed an increase and subsequent decrease, the triglyceride levels remained flat throughout the test in both groups. Free fatty acid levels were suppressed compared to baseline during the test, but PL patients had significantly higher FFA from time 30 to time 180 ($p < 0.05$) and tended to suppress less. While controlling for the differences in glucose levels, GIP levels displayed a large peak at time 30 min in both groups but were significantly higher over the course of the test in the PL group (AUC: 32542, pg/mL x min (20528-57728) vs. 3343 pg/mL x min (1728-4498), $p < 0.05$). In contrast, GLP-1 levels (also peaking at time 30 min in both groups), were significantly lower in PL throughout the test (AUC: 3017 pg/mL x min (2309-6051) vs. 28387 pg/mL x min (20422-36045), $p < 0.05$). Ghrelin and PPY levels did not differ significantly between the two groups. **Interpretation/Conclusion:** PL patients displayed more profound hyperglycemia and impaired suppression of FFAs. Interestingly, PL patients did not show substantial increases in triglyceride levels during MMT. There was a striking difference in the incretin responses between the two populations despite controlling for glucose, suggesting that MMT may have a role in differential diagnosis PL. Also, altered incretin response should be investigated as a contributor to metabolic perturbations and pathophysiology of PL.

Steroid Hormones and Receptors

STEROID AND NUCLEAR RECEPTORS

Regulatory Sharing between Estrogen Receptor Alpha Bound Enhancers

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Mammalian genomes encode an order of magnitude more gene expression enhancers than promoters, suggesting that