in the SR detected with Fluo-5N, a specific SR calcium indicator, and caused VSMC relaxation. These effects were blocked by cyclopiazonic acid (CPA, a SERCA inhibitor), suggesting that SERCA plays a critical role in P4 induction of VSMC relaxation. Similarly, the effects of P4 and OD 02-0 on relaxation of umbilical artery rings measured with a myograph were significantly attenuated by CPA, which confirms the critical role of SERCA in the rapid action of P4 and 02-0 on vascular muscle relaxation. P4 has previously been shown to activate MAPK and Akt signaling pathways to induce VSMC relaxation. The P4- and OD 02-0-induced increases in calcium in the SR were blocked by MAPK and Akt/Pi3k signaling inhibitors, AZD6244 and wortmannin. Taken together, these results suggest that the direct, rapid effects of P4 on relaxation of VSMCs through mPRα involves regulation of the expression and function of the SR proteins SERCA and PLB through MAPK and Akt signaling pathways.

Diabetes Mellitus and Glucose Metabolism

DIABETES COMPLICATIONS II

Dulaglutide Commonly Known as Trulicity; An Anti-Diabetic Medication Causing Small Bowel Obstruction

Ankita Gandhi, MD[1], Verisha Khanam, MD[1], Daymon Peterson, MD[1], Fenil Patel, MD[2], Zain Kulairi, MD[1].
[1]Wayne State University, Rochester, MI, USA, [2]Center for Family Medicine, Sioux Falls, SD, USA.

MON-681

Introduction Small bowel obstruction is a common and life threatening surgical emergency. The general causes are intra and extra-intestinal mechanical obstruction, such as from post-operative adhesions, malignancy, hernias, Crohn's disease, and volvulus. Less frequently neurologic, metabolic, and medications interfere with intestinal motility and lead to obstructive features. Here, we present a rare case of small intestinal obstruction caused by the anti-diabetic glucagon-like peptide 1 (GLP-1) agonist, Dulaglutide (Trulicity). **Case** A 52-year-old male with Diabetes Mellitus, presented with two weeks of severe nausea and vomiting, accompanied by four days of diffused abdominal pain. CT scan of the abdomen showed multiple mildly distended dilated loops of the proximal jejunum. The results lead to suspect the presence of fecal stasis with an apparent transition zone of a normal caliber bowel. This is strongly indicative of partial or evolving small bowel obstruction. The patient was treated with conservative management of bowel rest and NG tube per the surgery on board. However, patient deteriorated too quickly and the partial bowel obstruction lead to full obstruction and eventually taken for an emergent surgery. With careful investigation to identify underlying causes of small bowel obstruction revealed no mechanical, structural, or metabolic explanation. However, a review of patient's medication list disclosed a daily consumption of Dulaglutide (Trulicity). The medication was started 3 weeks prior to admission. He started developing partial bowel obstruction symptoms within one week of starting the medications. Unfortunately, surgeon ended up performing a partial resection a small bowel due to severe ischemia. Patient improved clinically in four to five days and was discharged home with an alternative anti-diabatic medications. He follows up with us in the clinic and has no signs of bowel obstructions with the other anti-diabatic medications. **Discussion:** Dulaglutide (Trulicity) is associated with small bowel obstruction. The side effect is more common in males and in patients who are using the medication for less than one month. A total of 8 cases were reported in 2017 with majority of them requiring surgical intervention for the small bowel obstruction. In our patient, it also required a surgical intervention and was life threatening. Unfortunately, the actual mechanism Trulicity causing the small bowel obstruction is unknown; however the moderate side effect of Trulicity is constipation. In this case, our patient was not constipated. He had normal bowel movements on a regular basis. Also, he never had any history of abdominal surgeries which can cause adhesion and lead to small bowel obstruction. All the other caused of small bowel obstructions had been ruled out and finally concluded Trulicity was the culprit of this unfortunate case.

Bone and Mineral Metabolism

PARATHYROID HORMONE TRANSLATIONAL AND CLINICAL ASPECTS

The Role of β-arrestin2 in Bone Catabolic Response to Hyperparathyroidism In Vivo

Gilberto Li Feng, BSc[1], Marc Grynpas, PhD[2], Jane Mitchell, PhD[1].
[1]University of Toronto, Toronto, ON, Canada, [2]Lunenfeld-Tanenbaum Research Institute, Mount Sinai Hospital, Toronto, ON, Canada.

SAT-392

Primary hyperparathyroidism (PHPT) is an endocrine disorder characterized by elevated parathyroid hormone (PTH) levels and hypercalcemia caused by the overactive parathyroid glands, resulting in negative impacts on the skeleton including bone loss and increased bone fragility[1]. PTH binds and activates parathyroid hormone type 1 receptor (PTH1R) which primary couples to Gαs, stimulating the downstream effectors that mediate bone remodeling processes[2]. PTH1R activity is regulated by arrestins, specially β-arrestin2 (β-arr2), through signal termination and receptor internalization[2]. Previously, we have seen anabolic effects of hyperparathyroidism (cPTH) on trabecular bone in mice overexpressing Gαs[3]. We hypothesized that increased Gαs protein levels in osteoblasts outcompete β-arr binding to PTH1R, leading to reduced signal termination and increased bone formation. To test this hypothesis, we are testing if the deletion of β-arr2 will also result in an anabolic response to cPTH in this study. The response of β-arr2 knockout (KO) mice to cPTH have yet to be documented. The hypothesis of this study is that β-arr2 KO mice treated with cPTH will exhibit anabolic effects on the trabecular bone. Nine-week-old wild-type (WT) C57BL/6 and β-arr2 KO mice were treated for 14 days with either rPTH1-34 (80ng/g/day) or saline (PBS) using micro-osmotic pumps to simulate hyperparathyroidism. There are 8 groups (n=10 per group) including both sexes, 2 genotypes (WT and KO), and 2 treatment groups (PTH and PBS). Two 30 mg/kg doses of 0.6% calcein green were administered subcutaneously to