a somatostatin receptor scintigraphy study was done which showed evidence of a somatostatin avid receptor rich lesion in the lung, corresponding to the known tumour. Thus, what initially appeared to be a SCLC was subsequently found to be a GHRH-secreting NET, illustrating the challenges of diagnosing these rare tumours. She was treated with intramuscular sandostatin 20mg q28days and then underwent a left pneumonectomy, resulting in biochemical resolution of her acromegaly (IGF-1 219 μg/L). Genetic testing for MEN1 was also completed as there is an association between ectopic GHRH NETs and MEN-1; however, genetic testing revealed no pathogenic variants and exon deletions or duplications suggestive of MEN-1.
Learning points
Diagnosis of acromegaly due to ectopic GHRH secretion requires high clinical suspicion by the treating clinicians (e.g., endocrinologist, oncologist) as well as review of histology by expert pathologists. Our case highlights that ectopic GHRH secretion is a rare but important cause of acromegaly, which should always be suspected when a clear pituitary cause is not identified.

Cardiovascular Endocrinology

HYPERTRIGLYCERIDEMIA; INFLAMMATION AND MUSCLE METABOLISM IN OBESITY AND WEIGHT LOSS I

Should Severe Hypertriglyceridemia Also Be Considered as a Contraindication for Use of Glucagon like Peptide 1 (GLP-1) Agonists?

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SAT-570

Background: Reported cases of acute pancreatitis have been associated with the use of GLP-1 agonists for treatment of diabetes mellitus. Hypertriglyceridemia is a well-established but underestimated cause of acute and recurrent pancreatitis. At the present time, there is insufficient data to know if there is a casual relationship. **Clinical Case:** A 46 y.o. male with past medical history of coronary artery disease, hyperlipidemia, type 2 diabetes mellitus, hypertension, and morbid obesity, was admitted to the hospital with severe abdominal pain radiating to the back associated with non-bilious vomiting, for 1 day. Patient endorsed that 4 years ago he was diagnosed with hypertriglyceridemia. Physical exam findings were notable for a distended abdomen with mild epigastric tenderness, heart rate at 120 bpm, and a body mass index of 37 kg/m^2. Active medications include: atorvastatin 40 mg PO daily, fenofibrate 45 mg PO daily, metformin 1,000 mg PO twice a day, glipizide 5 mg PO daily, levemir 60 units SQ twice a day, and most recently he had been started on dulaglutide 0.75 mg SQ weekly. Initial tests were consistent with acute pancreatitis and diabetic ketoacidosis: lipase 944 U/L (n 8.0 - 78 U/L), anion gap 18 mEq/L (n 5 - 13 mEq/L), creatinine 1.6 mg/dL (n 0.72 - 1.25 mg/dL), glucose 479 (n 60–100 mg/dL), β-Hydroxybutyrate 5.3 mmol/L (n <0.3mmol/L), urine glucose >1,000 mg/dL (n Negative mg/dL), urine ketones 20 mg/dL (n Negative), Triglycerides (TG) 5,374 mg/dL (n <150 mg/dL) and Hgb A1C 11.9% (n <5.7%). CT abdomen and pelvis without contrast revealed moderate acute pancreatitis. Patient was admitted to the intensive care unit and was started on intravenous insulin, atorvastatin 80 mg PO daily and fenofibrate 145 mg PO daily. Despite optimization of lipid-lowering agents, TG remained above 2,000 mg/dL. Decision was made to start patient on plasmapheresis until TG was <500 mg/dL. Patient's TG improved to 370 mg/dL after second treatment. Patient's dulaglutide was discontinued and patient was advised to avoid GLP-1 agonist use, indefinitely. One-month post discharge patient's TG level was 370 mg/dL. **Conclusion:** Pancreatitis should be considered in patients on GLP-1 agonists, that present with persistent severe abdominal pain (with or without nausea), and its use should be discontinued in such patients. Use of GLP-1 agonists should be avoided in subjects with severe hypertriglyceridemia. Further research should be made in order to determine if GLP-1 agonists should be contraindicated in patients with severe hypertriglyceridemia, as both increase risk for pancreatitis.

Bone and Mineral Metabolism

BONE AND MINERAL CASE REPORTS I

Diagnosis of Autosomal Dominant Hypocalcemia Type 1 Following the Initiation of Imatinib for Treatment of Chronic Myeloid Leukemia

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SAT-347

Background: Autosomal Dominant Hypocalcemia Type 1 is an underdiagnosed condition due to the vast majority of patients being asymptomatic and having mild hypocalcemia. Imatinib has been associated with hypophosphatemia and hypocalcemia.
Clinical Case: 69-year-old man who had a long history of asymptomatic mild hypocalcemia, calcium level of 7.9 mg/dL (8.4 – 10.2 mg/dL) diagnosed with chronic myeloid leukemia and developed symptoms of hypocalcemia within 2 months of treatment with imatinib. After the initiation of imatinib, his calcium reduced to 6.8 mg/dL (8.4 – 10.2 mg/dL) with a corresponding ionized calcium of 0.98 mmol/L (1.12–1.32 mmol/L) within 2 months. He developed tetany. With the reduced calcium level his PTH was unexpectedly low-normal at 34 pg/mL (16–62 pg/mL). He also had a higher than expected urinary calcium of 342 mg/24 hours. The PTH and 24-hour urinary calcium levels raised concern for an underlying diagnosis of autosomal dominant hypocalcemia type 1. His phosphorous was normal at 3.3 mg/dL (2.6–4.9 mg/dL). He never had hypophosphatemia, which is common with imatinib.
After two doses of IV calcium and initiation of oral calcium replacement, 1000 mg BID, his level continued to be reduced with symptoms. Given his symptoms, laboratory results, and continued hypocalcemia he underwent genetic testing. Results of his genetic testing showed a p.Thr151Met mutation in the CASR gene consistent with autosomal dominant hypocalcemia type 1. With this diagnosis, there is concern for nephrolithiasis with over treatment. His symptoms resolved with treatment with calcium 1000mg TID, calcitriol 0.25 mg BID and vitamin D3 2000 IU daily. He has not developed nephrolithiasis and his urinary calcium increased

to 424mg/24 hours. A thiazide diuretic is being considered to decrease urinary calcium excretion.
He continues on imatinib with an excellent response to therapy. It is likely that the trigger for symptomatic severe hypocalcemia was initiation of imatinib superimposed on the hypocalcemia type 1. Possible mechanisms for this are decreased intestinal absorption, increased urinary loss, or decreased dissolution from bone. [1] These mechanisms are also associated with hypophosphatemia, which our patient did not have. Another possible cause could be an immune mediated destruction of the parathyroid gland, a rare finding seen with other tyrosine kinase inhibitors.[2]
Conclusion: This is the first reported case of imatinib triggering severe symptomatic hypocalcemia leading to the diagnosis of underlying autosomal dominant hypocalcemia type 1.
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Thyroid

THYROID DISORDERS CASE REPORTS I

Treatment of Uncontrolled Graves' Disease Reveals Underlying Thyroiditis

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SUN-492
Thyrotoxicosis is a life-threatening condition that may result from poor adherence to medical therapy. We describe the case of a patient with Graves' disease and a history of medication non-compliance who presented with tachyarrhythmia and heart failure (HF). His protracted course after weeks of inpatient treatment with thionamides, iodine, resin and beta-blockers, was followed by a dramatic improvement within days of adding steroids, suggesting the presence of concurrent untreated thyroiditis.
Case Description: A 38-year-old man with an 18-month history of known Graves' disease presented to the endocrinology clinic requesting thyroidectomy. He complained of 3 weeks of worsening palpitations, shortness of breath, bilateral lower extremity edema, and frequent loose stools. History revealed frequent interruption of thionamide and recent discontinuation of propranolol due to patient's perceived lack of improvement. Physical exam showed a thin male with a large goiter. He was in moderate distress due to tachypnea. He had atrial fibrillation with rapid ventricular rate (A Fib with RVR) but was not in pulmonary edema. Laboratories showed a suppressed TSH (<0.02 mIU/L [0.45 - 4.70 mIU/L]), a free T4 above the level of detection (>6.99 ng/dL [0.78 - 2.20 ng/dL], and a total T3 level of above 781 pg/mL (97.0 - 170.0 ng/dL). Methimazole 30 mg BID and propranolol 40 mg TID were started, followed within a few hours with potassium iodine (SSKI) 3 drops TID. Although the patient's HF improved and there was down-trending of free T4, he continued to have A fib with RVR. SSKI and propranolol were increased; diltiazem 90 mg QID and cholestyramine 2g BID were later added. Despite aggressive treatment, the patient continued to have intermittent tachyarrhythmia, postponing his planned thyroidectomy for weeks. Dexamethasone 2mg Q8H was then started aiming to further decrease T4 to T3 conversion. Interestingly, within 48 hours of the start of steroid therapy, his free T4 level markedly decreased and was well within the normal range several days later when he underwent a successful thyroidectomy, with no further tachyarrhythmia recurrence.
Discussion: Thyrotoxicosis is aggressively treated given its high mortality rate. Treatment algorithms guide physicians to categorize hyperthyroidism either as the type associated with increased hormone synthesis and secretion or that which results from increased hormone release due to gland destruction. This thought process may lead, as in our case, to a delay in appropriate therapy and an increased risk of disease complications. Furthermore, providers should not assume that the lack of treatment response in a patient with Graves' disease is the result of medication nonadherence. The possibility of coexisting thyroiditis and the absence of a treatment regimen that effectively addresses both underlying processes may be the actual cause for the lack of clinical improvement.

Diabetes Mellitus and Glucose Metabolism

PREGNANCY, LIPIDS, AND CV RISK — IMPACT OF DIABETES ACROSS THE SPECTRUM

Do OGTT-based Insulin Secretory Response Measures Approximate 1st Phase Insulin Response in Pregnant Women?

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OR08-02
Background: We previously showed that 1st phase insulin response increases dramatically in pregnancy, independent of changes in insulin sensitivity. Measurement of 1st phase insulin response requires the use of hyperglycemic clamps or intravenous glucose tolerance tests (IVGTTs) which are rarely performed in pregnant women. Oral glucose tolerance test (OGTT)-based measures of insulin secretory response have not been validated in pregnancy.
Methods: In a secondary analysis of a longitudinal study of glucose metabolism in pregnancy, we examined Pearson correlations between OGTT-based insulin secretory response measures and 1st phase insulin response. Forty women were studied pre-pregnancy; 36 returned in early and late pregnancy (12–14 and 34–36 weeks gestation). At each time point, after overnight fasts, an IVGTT and an OGTT were performed on separate days. The 1st phase insulin response was calculated as the incremental area under the curve during the 1st 10 minutes after intravenous administration of a 0.5 g/kg glucose load (or 19g/m^2 body surface area if