SAT-410  
Background Radioactive iodine therapy (RAI) is an excellent choice to treat thyrotoxicosis, particularly Graves’ disease (GD) patients. After RAI therapy, it is well known that TSH receptor antibodies (TRAb) rise in GD patients and autoimmunity can eventually surge in patients with toxic multinodular or uninodular goiter (TNG). Recently, biological assay distinguishes stimulating TRAb, called thyroid stimulating immunoglobulin (TSI) bringing a new perspective on persistent thyrotoxicosis and ophthalmopathy after RAI therapy.  
Objective Analyze TRAb and TSI levels after 6 and 12 months of RAI therapy for thyrotoxicosis. Patients and Methods Patients were evaluated prospectively immediately before and 6 to 12 months after RAI therapy for thyrotoxicosis. Thyroid hormones were all measured using immunoassays (Roche Diagnostics Ltd). TRAb was analyzed by Elecsys Anti-TSHR assay (Roche Diagnostics, Germany) and was considered negative if < 1.75 IU/L (analytical range: 0.3 to 40 IU/L). TSI was measured by Immulite TSI assay (Siemens Healthcare, UK) and was considered negative if < 0.55 IU/L (analytical range: 0.1 to 40 IU/L). Clinical data and comparison of assays were analyzed by SPSS and MedCalc softwares. Results From 2017 to 2019, 54 patients (44 females) were prospectively evaluated after 6 months of RAI therapy, mostly because of GD (40 patients). A high degree correlation was observed between TRAb and TSI (Spearman correlation coefficient =0.875; p < 0.0001, 95% CI 0.784 to 0.929). After 6 months, among patients with GD, 5/40 patients had negative TRAb levels and 2/40 had negative TSI levels, whereas all TNG patients had both negative TRAb and TSI levels. In GD group, 4 patients showed subclinical hyperthyroidism and relapse occurred in 1 case. All patients with TNG showed euthyroidism status with or without thyroid medications. One year after RAI therapy, we evaluated 32 patients (23 GD) and 4/23 of GD had negative TRAb levels and only 1/23 had negative TSI level. All patients with TNG had negative TRAb and TSI levels after one year treatment. Subclinical hyperthyroidism was diagnosed in 5 patients with GD but none with TNG. Along follow-up, 4 patients with clinical diagnosis of GD with TRAb negative before RAI therapy became positive after RAI therapy and 3 patients became TSI positive. Conclusions Long term after RAI therapy for thyrotoxicosis treatment, TRAb and TSI are still positive in most GD patients and few cases can even turned to positive levels. Nevertheless, in TNG patients, RAI therapy is safe as TRAb and TSI maintained at negative concentrations and thyrotoxicosis is properly resolved.

BONE AND MINERAL METABOLISM  
CLINICAL ASPECTS OF OSTEOPOOROSIS AND VITAMIN D ACTION  
Total and Free 1,25-dihydroxyvitamin D Levels in Postmenopausal Patients with Primary Hyperparathyroidism  
Lingqiong Meng, MD, MS, Sue A. Shapses, PhD, Xiangbing Wang, MD, PhD.  
Rutgers University, New Brunswick, NJ, USA.
is 1-5% in patients treated with 131I therapy for hyperthyroidism. Dose of RAI and thyroid volume can be precipitating factors for post radiation thyroiditis. The higher RAI dose increases the chance of RAI thyroiditis while the larger goiter size decreases the absorbed radiation dose in the thyroid gland. We present a 25-year-old Emirati male previously healthy, who was referred to our service for hyperthyroidism management. He presented with thyrotoxicosis in absence of goiter. All investigations revealed that Graves’ disease is the primary cause of his hyperthyroidism. He was treated with RAI ablation 18.3 mCi. Day four after RAI, he presented with severe pain in the anterior neck associated with fatigue, tremor, palpitation and weight loss. Symptoms lasted for 6 weeks post RAI. There was laboratory evidence of thyrotoxicosis presented with further suppression of TSH and higher FT4 than the baseline. Acute radiation thyroiditis was diagnosed and has been commenced on propranolol 10mg BID. Symptoms completely resolved after 6 weeks of treatment and thyroid function returned to normal level. The patient has remained asymptomatic on continued follow up care till eventually became hypothyroid clinically and biochemically. Our case reflects that radiiodine thyroiditis can last for longer period and occur after larger doses of 131I treatment in absence of goiter. Our patient has RAI induced thyroiditis lasted for 6 weeks post 18.3mCi of 131I, and has no goiter, which were both contributing factors for RAI induced thyroiditis.

Diabetes Mellitus and Glucose Metabolism

TYPE 1 DIABETES MELLITUS

Pembrolizumab Induced Worsening Glycemic Control and DKA in Type 2 Diabetes Mellitus.

Sarita Goud, MD, Yu Yu Thor, MD.
Mercy Catholic Medical center, Darby, PA, USA.

SAT-676

INTRODUCTION

Pembrolizumab (Keytruda) is a humanized IgG4 anti-programmed cell death-1 (PD-1) antibody serving as an immune-checkpoint inhibitor, now approved by FDA to treat several types of cancer. Although there are few reported cases of pembrolizumab induced new onset DKA in a non diabetic patients due to its autoimmune nature, its association in worsening glycemic control and DKA in pre-existing type 2 Diabetes mellitus is not well established.

CASE

79 years old female with past medical hx of DM type 2 (Hba1c 7.4 was started on metformin), COPD(on chronic steroids and trilogy machine at home), recently diagnosed with poorly differentiated adenocarcinoma of the left lung, metastasis to liver, PDL 1 positive at 99%, started on palliative chemotherapy with Keytruda, 2 weeks after the third cycle of keytruda presented to the ED for AMS. Patient noted to be very dehydrated, somnolence and tachypnea. Labs consistent with sugars > 600, potassium 6.8, Bicarb 5, Anion gap 33, beta hydroxybutyrate 11.5 (on 7/15/19 0.6), HbA1c 9.7,(On 12/15/16 7.3, 9/25/18 6.7, 1/22/19 7.4), PH 7.31, lactate 2.4. WBC count 21.5- no infectious source identified (CXR, CT brain, UA clean). Patient was admitted for DKA and treated with IV insulin and IV fluids. After medically stable patient was discharged with Insulin regimen. Within 5 days after being discharged, patient presented to ED again with DKA with PH 7.27, Bicarb 8, anion gap 22, sugars>600, beta hydroxybuterate 13.70. Patient was Rx for DKA- after a week of hospitalization was discharged to Hospice(due to metastatic cancer) and few weeks later expired.To summarize, pt with well controlled type 2 DM on metformin presented with frequent DKA 2 weeks after treatment with third cycle of keytruda leading to worsening glycemic control in-turn making patient insulin dependent.

CONCLUSION

Incidence of Type 1 DM with pembrolizumab treatment is being increasingly recognized and reported, and DKA is a common initial presentation. However we need further studies to establish the mechanism of worsening glycemic control leading to Insulin dependent and DKA in patients with pre-existing type 2 diabetes. Also, physicians should counsel patients about this potential immune related adverse effect and educate them about the symptoms of hyperglycemia and DKA.

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Adrenal

ADRENAL - CORTISOL EXCESS AND DEFICIENCIES

Diagnostic Utility of Free Cortisol in Dexamethasone Suppression Test. A Prospective Study in Healthy Subjects.

Shobana Athimulam, MD, Ravinder Jeet Kaur, MBBS, Ravinder Ji Singh, PHD, Stefan Karl Gunther Grebe, PHD, MD, Irina Bancos, MD.
Mayo Clinic, Rochester, MN, USA.

MON-169

OBJECTIVE: Low dose dexamethasone suppression testing (DST) is standard of care in patients with adrenal incidentalomas or suspected endogenous hypercortisolism. Pulse positive total serum cortisol (TC) results occur due to poor absorption, rapid metabolism or estrogen use. Free serum cortisol (FC) measurement is an alternative, but optimal cut-offs are unknown. We aimed to establish the optimal serum dexamethasone concentrations (DEXA) to interpret TC and FC results and identify reasons for discrepancies between TC and FC values in healthy male and female (with and without oral contraceptive therapy, OCT) subjects. METHODS: Single center prospective study of healthy subjects >17 years old undergoing assessment with DST between 2016 and 2019. Measurement of FC and DEXA was performed by tandem mass spectrometry and