Adrenal

ADRENAL CASE REPORTS I

Bilateral Adrenal Hemorrhage with Clinically Preserved Adrenal Function Leading to the Diagnosis of Antiphospholipid Syndrome.

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SAT-182

Background: Spontaneous bilateral adrenal hemorrhage (BAH) is a rare complication of antiphospholipid syndrome (APS), which is the most common identifiable risk factor for BAH. Although adrenal dysfunction is generally irreversible, adrenal function might be preserved or even recover in rare cases[1].

Clinical case: A 48 year-old man with history of hypertension and gout presented with right upper quadrant abdominal pain following trauma to his left leg. He was found to have a left lower extremity deep vein thrombosis and bilateral pulmonary emboli (PE) and was started on anticoagulation therapy. He continued to have abdominal pain and a CT abdomen revealed BAH. Three am cortisol level was 21 mcg/dL (8–25 mcg/dL), ACTH 37 pg/mL (6–59 pg/mL), aldosterone <3 ng/dL (4–31 ng/dL), renin 2.6 ng/mL/hr (0.2–1.6 ng/mL/hr), sodium 130 mmol/L (135–146 mmol/L) and potassium 4.3 mmol/L (3.6–5.3 mmol/L). Patient was hemodynamically stable and did not report symptoms of adrenal insufficiency. Hypercoagulable work-up was consistent with APS and Lupus. Despite normal cortisol levels, he was started on hydrocortisone in the setting of anticoagulation and recent hemorrhage. Given low aldosterone with slightly high renin he was also started on fludrocortisone. Six weeks after discharge, his morning cortisol was 6 mcg/dL and ACTH was elevated at 76 pg/mL which was concerning for adrenal insufficiency. However, 250 mcg IM ACTH stimulation test showed peak cortisol of 17 mcg/dL which is considered adequate. Aldosterone and renin levels normalized so fludrocortisone was discontinued.

Patient subsequently self-discontinued all medications for 1 month with no symptoms of adrenal insufficiency, and later restarted hydrocortisone on his own. Repeat ACTH stimulation test showed baseline ACTH 57 pg/mL with peak cortisol of 17 mcg/dL. Patient was tapered off hydrocortisone and displayed no subsequent symptoms of adrenal insufficiency.

Conclusion: This case highlights the need to consider APS in patients with spontaneous BAH. Additionally, patients with BAH may have relatively preserved adrenal function. There is limited data to guide when steroid replacement is necessary for patients without clear adrenal insufficiency. It may be reasonable to monitor these patients off hydrocortisone replacement with close monitoring.

1. Ramon I, Mathian A, Bachelot A, et al. Primary adrenal insufficiency due to bilateral adrenal hemorrhage-adrenal infarction in the antiphospholipid syndrome: long-term outcome of 16 patients. *J Clin Endocrinol Metab*. 2013;98(8):3179–3189.

Diabetes Mellitus and Glucose Metabolism

DIABETES DIAGNOSIS, TREATMENT AND COMPLICATIONS

Survival of Patients with Gastroenteropancreatic Neuroendocrine Tumors and Diabetes Mellitus in a Seer-Medicare Cohort

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SUN-626

Background: The incidence and prevalence of gastroenteropancreatic neuroendocrine tumors (GEP-NET) is increasing globally and has been associated with diabetes mellitus (DM). In this study we aimed to compare tumor characteristics, disease-specific survival (DSS) and overall survival (OS) of GEP-NET patients (pts) with and without DM. *Methods:* Using the Surveillance, Epidemiology, and End Results registry (SEER) linked to Medicare claims, we identified pts diagnosed with GEP-NET between January 1995 and December 2010, aged ≥65 years at the time of GEP-NET diagnosis. We included patients who were in exclusive Medicare coverage without healthcare management organizations and had Medicare Parts A and B coverage for ≥1year after GEP-NET diagnosis or until death. Within the pts with GEP-NET diagnosis, we identified those without a diagnosis of DM prior to the GEP-NET diagnosis. We compared baseline sociodemographics, co-morbidities, and GEP-NET location, stage, grade and treatment between pts with and without DM using χ^2 analysis. Kaplan Meier (KM) curves were used to compare OS and DSS up to 10 years between the DM and non-DM groups. We used Cox proportional hazards analysis to compare the DSS between the groups, adjusting for confounding variables. *Results:* We identified a cohort of 1,969 well-characterized GEP-NET patients with accurate tumor stage, grade, comorbidities, and treatment data. 478 (25.7%) had DM and 1,383 (74.3%) did not have DM. There were no statistically significant differences in gender or age at the time of GEP-NET diagnosis in the DM (mean age 74.7±SD 6.6 yrs) and non-DM (74.9±7.4 yrs) groups. Significant differences in race were found in the DM (80.6% white, 13.6% black, 1.3% hispanic) and non-DM (86.8% white, 8.2% black, 1.8% Hispanic) groups (p=0.002). Patients with DM had more gastric (14.7%), duodenal (10.9%) and pancreatic (21.0%), and less jejunal/ ileal (12.8%) NETs compared with the non-DM group (9.7%, 6.4%, 16.9%, 18.2%, respectively, p<0.0001). Patients with DM had earlier stage disease than those without DM (p=0.0012), but no difference in tumor grade or treatment was found. KM curves revealed no differences in OS and DSS in the GEP-NET patients with and without DM across all stages. Multivariate adjusted Cox proportional-hazards model found no significant difference in DSS between those with and without DM (HR=0.97, 95%CI: 0.76–1.24). Compared with pts with pancreatic NETs, pts with colon (HR=1.39, 95%CI: 1.04–1.86) had worse survival, while those with jejunal/ileal (HR = 0.59, 95%CI: 0.42–0.83) NETs had a better survival. *Discussion:* This is the first study to investigate the effect of DM on

survival of pts GEP-NETs. We found a high prevalence of pre-existing DM in pts with GEP-NETs, but no difference in OS or DSS in pts with and without DM. Interestingly, pts with DM had more foregut GEP-NETs which may suggest mechanistic links between DM and GEP-NETs at these sites.

Bone and Mineral Metabolism

BONE AND MINERAL CASE REPORTS I

New Onset Adult Idiopathic Primary Hypoparathyroidism Concomitant with Severe Aplastic Anemia

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SAT-335

Introduction: Primary hypoparathyroidism is a relatively rare cause of hypocalcemia with cases of primary hypoparathyroidism in the US estimated at 24–37 per 100,000 with 75% being due to neck surgery and 25% due to non-surgical causes. The clinical presentation depends on the acuity of development of hypocalcemia and the absolute level of serum calcium. Here is a case of severe hypocalcemia secondary to hypoparathyroidism of unknown etiology followed by the development of severe aplastic anemia.**Case report:** A 60-year-old Caucasian male presented to our ED with fatigue, tingling, numbness in extremities and was found to have severe hypocalcemia at 6.8 mg/dl and decreased PTH at 11 pg/mL. Calcium levels 8 months prior to presentation were normal. No history of neck surgery, radiation exposure or family history of autoimmune disorders. Initial workup included creatinine, magnesium and TSH were normal. Autoimmune panel (including PTH Abs and CaSR Abs), HIV test, hepatitis panel, serum protein electrophoresis were also negative. Infiltrative causes of hypoparathyroidism including hemochromatosis, malignancy and granulomatous diseases like sarcoidosis were ruled out with tissue sampling and lab workup. Sestamibi scan obtained showed no parathyroid activity in all four glands. Patient was initially treated with IV calcium to improve serum calcium to more than 7.5mg/L and then switched to oral calcium carbonate 500mg TID and calcitriol 0.5mcg BID until the calcium level was brought up to the lower limit of normal. Patient was seen in follow up and was doing well without any adverse effects. Consequently, the patient developed severe aplastic anemia which was treated with steroids and interestingly, has caused a gradual but consistent increase in PTH levels.**Discussion:** Idiopathic hypoparathyroidism (IHP) is a rare condition with an incidence of 0.02%. IHP can occur sporadically or as part of a familial condition with autosomal dominant, recessive and X-linked recessive patterns. Certain autosomal forms of hypoparathyroidism have mutations in the PTH gene and Calcium-Sensing Receptor (CaSR) gene. The challenging nature of this case is due to the subacute nature of the patient's presentation along with the lack of a definitive etiology. The patient's negative family history and older age makes genetic causes less likely, and Abs against PTH and CaSr were also negative. The patient's diagnosis of severe aplastic anemia has made the case more fascinating, especially since its management with steroids has causes an improvement in the patient's PTH status. Regardless of etiology, primary hypoparathyroidism is treated with lifelong supplementation of calcium and calcitriol to a goal serum calcium level at the lower limit of normal. **Reference:** Abate EG,Clarke BL. Review of Hypoparathyroidism. Front Endocrinol (Lausanne). 2017; 7:172. Published 2017 Jan 16

Thyroid

THYROID CANCER CASE REPORTS II

Tall Cell Variant of Papillary Thyroid Carcinoma Presenting Acutely as a Right Thyroid Mass and Thyroiditis - an Unusual Presentation

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MON-442

INTRODUCTION

Aggressive malignant thyroid tumors (AMTT) can rarely present with signs and symptoms mimicking that of acute suppurative thyroiditis (AST). In its early course, correct diagnosis may be difficult but a prompt recognition is crucial. We report an interesting case of an aggressive papillary thyroid cancer with presenting acute symptoms masquerading as AST.

CLINICAL CASE

A 50-year-old Caucasian male was admitted for 2 day history of sore throat, odynophagia, low grade fever with a tender and enlarging right neck swelling, not improving with oral antibiotics. CT neck showed a 5.8 x 4.5cm mass in right thyroid lobe with shallow retropharyngeal effusion. Labs showed elevated WBC 10.7 x 10^9/L (4–10.5), ESR 20 mm/hr (0–12), CRP 92.93 mg/L (0.2–10), with negative rapid strep and IgM for viruses EBV and CMV. Thyroid function tests were normal. Ultrasound neck showed an ill-defined 6.5 cm hypoechoic mass with cystic component and increased vascularity and calcification, replacing the right thyroid lobe. Due to concern for thyroid abscess he underwent urgent aspiration. Fluid analysis and cultures were negative for bacterial source. FNA (fine needle aspiration) of one specimen was suspicious for follicular neoplasm with predominant hurtle cells and suspicious for thyroid neoplasm with papillary architecture in the other. Core biopsy was suspicious for papillary carcinoma but limited due to scant amount of tissue. Affirma unavailable due to insufficient RNA yield. Flow cytometry was negative for abnormal lymphocyte population. Repeat CT neck 3 weeks later was concerning for metastatic lymphadenopathy. He underwent total thyroidectomy with lymph node (LN) dissection and pathology showed papillary thyroid cancer, tall cell variant, tumor size 6 cm, predominantly hemorrhagic, extending to perithyroidal strap muscles and involving 5/6 LN in right trachea-esophageal groove. It was a stage I, pT3bN1aMx cancer. Subsequently, he underwent thyrogen stimulated I-131 ablation of residual thyroid carcinoma with pretherapy scan showing increased uptake in thyroid bed and a satellite lesion suspicious for LN metastasis. Post-ablative scan showed no other new lesions. He is now on levothyroxine suppressive therapy.