guidelines, as mild, moderate, or severe. The child's history prior to diagnosis was recorded, and patients underwent a number of clinical assessments, including anthropometry, biochemical parameters, and OGTT. The demographic and clinical characteristics of children with and without DKA were compared. **Results:** 681 patients (314 boys and 367 girls) were diagnosed with T1D over the study period. 341 (50.1%) had DKA at diagnosis, with yearly rates ranging from 45.0% to 56.8%. Eight cases had missing data on severity, but 120 had mild (36.0%), 100 moderate (30.0%), and 113 severe (33.9%) DKA. Children with DKA were younger than those without DKA (7.2 vs 8.2 years, respectively; p=0.001), and were more likely to report vomiting (13.2% vs 1.5%; p<0.001), loss of appetite (3.2% vs 0.9%; p=0.031), fatigue (39.3% vs 5.6%; p<0.001), and abdominal pain (9.1% vs 1.5%; p<0.001) prior to T1D diagnosis. The classical symptoms of diabetes were similar in children with and without DKA, such as polyuria (86.8% vs 90.3%, respectively; p=0.153), polydipsia (90.6% vs 91.5%; p=0.696), polyphagia (26.4% vs 30.3%; p=0.259), and weight loss (50.7% vs 50.6%; p=0.970). Children with DKA had higher blood lipids and fasting insulin levels at presentation, but displayed similar HbA1c levels. **Conclusions:** We report a high incidence of DKA at our centre (50%) among children diagnosed with T1D. As most children displayed classical symptoms of diabetes, it is important to increase community awareness of the disease to ensure an early diagnosis and lower the risk of children presenting with DKA.

Bone and Mineral Metabolism

BONE AND MINERAL CASE REPORTS II

Treatment of Parathyroid Storm in a Patient with Traumatic Brain Injury: A Reversible Comatose State

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MON-370

Background:
Parathyroid storm, also known as parathyroid crisis, is a rare and under-recognized endocrine emergency due to severe hypercalcemia in patients with primary hyperparathyroidism. It is characterized by significantly elevated parathyroid hormone (PTH) levels even up to 20 times above the normal limit along with calcium levels >15 mg/dl, leading to multiorgan dysfunction, notably altered mental status and acute kidney injury. Risk of mortality is high without urgent parathyroidectomy. We describe a case of a patient with acute traumatic brain injury and parathyroid storm with PTH >1700 pg/ml (11-51) and Ca 15.4 mg/dl (8.6-10.4) in whom resection of a parathyroid adenoma reversed the comatose state.
Clinical Case:
Our patient is a 68 year-old male with no significant past medical history who sustained a fall off a 12-foot ladder complicated by right intracranial bleed s/p hemicraniectomy and multiple fractures, including left clavicle fracture with possible subclavian artery injury, left rib fractures, and right hip fracture s/p ORIF. The patient had a brief, partial improvement of mental status, followed by comatose state in the setting of rapidly rising calcium levels and acute kidney injury. In the setting of blood transfusions, the patient had an initial Ca of 8.8 mg/dl (8.6-10.4) on admission. The calcium levels rose over a week to 15.4 mg/dl with albumin of 2.4 g/dl (3.9-5.0), PTH levels from 953 pg/ml to >1700 pg/ml (11-51) after tracheostomy, and creatinine from 0.69 mg/dl to peak of 2.0 mg/dl (0.60-1.30). In spite of IV hydration, calcitonin, cinacalcet up to 90mg twice daily, pamidronate 60mg IV, and several sessions of hemodialysis, the patient's calcium did not normalize, and the patient remained comatose. Other labs showed phosphorus nadir of 1.4 mg/dl (2.3-4.4), 25-OH VitD 13 ng/ml (20-50), 1,25-OH VitD 9.8 pg/ml (19.9-79.3), VitA 0.6 mg/L (0.3-0.9), PTHrP <2.0 pmol/L (0.0-2.3), normal SPEP/UPEP, and peak CK of 569 U/L (63-474). Sestamibi scan showed intense tracer uptake within a nodule near the suprasternal notch, and parathyroid 4D-CT showed a left 17mm pretracheal lesion with cystic degeneration along the superior margin of the manubrium. The patient subsequently underwent parathyroidectomy of an ectopic cystic mass with normalization of calcium and PTH levels. Pathology revealed a 0.8 gram, 1.5 x 1.0 x 0.3 cm enlarged, hypercellular parathyroid. The patient woke up from his comatose state immediately after surgery with progressive improvement in mental status back to baseline, other than left-sided weakness.
Conclusion:
Our case highlights the importance of surgical management as an effective cure for parathyroid crisis and underscores the associated critical and significant rise in calcium and PTH levels, which was resistant to medical treatment.

Thyroid

THYROID NEOPLASIA AND CANCER

Primary Adrenal Insufficiency During Tyrosine Kinase Inhibitors Treatment in Advanced Thyroid Cancer Patients

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MON-537

Objective: Tyrosine kinase inhibitors (TKIs) are used for the treatment of metastatic differentiated (DTC), poorly differentiated (PDTC) and medullary (MTC) thyroid cancer. Several adverse events (AEs) have been reported in almost all patients (pts) treated with TKIs. One of the less known AE related to the use of these drugs is the primary adrenal insufficiency (PAI).
Methods: We analyzed the basal and stimulated adrenal function, ACTH levels, adrenal antibodies and electrolytes levels in 82 thyroid cancer pts treated with TKIs (vandetanib and cabozantinib in MTC pts, lenvatinib and sorafenib in DTC and PDTC pts) and we correlated these results with the clinical-pathological features of our pts.
Results: In our series, 25/82 (30.5%) pts showed a PAI after stimulation test with a progressive ACTH increase in 14/25 (56%) pts. Thirteen/25 (52%) pts with PAI were DTC, 8/25 (32%) pts were MTC and 4/25 (16%) pts were PDTC. Sixteen/25 (64%) pts were treated with lenvatinib, 8/25 (32%) were treated with vandetanib and 1/25 (4%) was

treated with cabozantinib at the time of stimulation test. In 5/25 (20%) pts PAI occurred within 12 months from the TKIs treatment initiation, in 9/25 (36%) within 36 months and in 11/25 (44%) after 36 months of treatment. Eighteen/25 pts with PAI were older than 55 years. Twenty/25 (80%) of these pts were treated with cortisone acetate replacement therapy with the improvement of fatigue in a small part of these while other 5 pts were untreated due to the mild degree of PAI and the absence of specific symptoms (i.e fatigue). Moreover, in our pts the evaluation of adrenal antibodies was negative and the electrolytes levels were in the normal range. We also correlated the presence of PAI with the clinical-pathological features of our pts but we didn't observe any significant correlation.

Conclusions: We observed that PAI, mainly subclinical, can occur during TKIs treatment in thyroid cancer pts. The appearance of fatigue, the typical symptom of PAI, could be multifactorial in these pts due also to the direct effect of TKIs treatment. Thus, in these cases is very difficult to recognize the cause of fatigue and to decide the appropriate treatment (cortisone acetate replacement therapy vs TKIs dose reduction). Moreover, the time of PAI appearance is variable since it can be early (<12 months) or late (>36 months) after TKIs treatment initiation and the adrenal function must be monitored during all TKIs treatment period. More studies are needed to know the pathophysiology of this "adverse event" during TKIs treatment and to improve the acknowledgments regarding the differential diagnosis and treatment of these pts, regardless of symptoms.

Genetics and Development (including Gene Regulation)

ENDOCRINE DISRUPTING CHEMICALS

Inhibition of Androgen Receptor Activity by DDE Is Affected by Mutations in the BF3 Site

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SAT-718

The androgen receptor (AR) plays an important role in the development of the male phenotype and traits. Some diphenyl compounds inhibit AR activity by binding to a hydrophobic surface binding site, BF3. A similar diphenyl structure is found in 4,4' DDT and its breakdown product 4,4' DDE. Previous results showed that DDT and DDE induced the release of bound dihydrotestosterone from the AR ligand binding domain, with IC_{50} values ranging from 54 to 82uM. This suggested that DDT and related compounds may act as endocrine disrupting chemicals by binding to the BF3 site and inducing allosteric changes in the AR structure, disrupting binding of the steroid to the ligand binding domain. Here, an AR reporter system was transiently transfected into HEK293 cells and AR activity was measured using a dual luciferase assay. The system was used to measure the response of the AR protein to varying concentrations of dihydrotestosterone in the presence and absence of DDE. DDE inhibited the activation of AR by dihydrotestosterone under these conditions. Five mutant AR genes with amino acid changes in the BF3 site were tested for alterations in the ability of DDE to disrupt AR activity. The five mutations tested were F673K, F673W, G724R, G724M, and L830D. The ability of DDE to inhibit AR activity was reduced by the mutations in the BF3 site. These results suggest that DDE acts as an endocrine disrupting chemical (EDC) by binding to the BF3 site and allosterically regulating AR activity.

Pediatric Endocrinology

PEDIATRIC GROWTH AND ADRENAL DISORDERS

Improved Adult Height in Brazilian Turner Syndrome Patients Treated with Growth Hormone

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SAT-107

Background: Short stature is the most frequent clinical manifestation in Turner syndrome (TS), occurring in 98% of these patients. Growth hormone was shown to improve adult height in TS patients from diverse genetic backgrounds. However, there are few studies on adult height in TS patients from developing countries, where the diagnosis is frequently delayed.

Objective: To compare adult height between GH-treated and untreated TS patients.

Patients and methods: 120 GH-treated and 109 GH-untreated TS patients from 3 referral hospitals in Brazil were evaluated. The most common reasons for not treating TS patients with GH were late diagnosis or GH unavailability. Data on karyotype, parents' height, puberty development and GH treatment were obtained from their medical records. Adult height was determined when growth velocity was inferior to 1cm/year during a minimum follow-up period of 12 months.

Results: The frequency of 45,X karyotype was similar between the groups (48.7% vs. 41.9% in GH-treated *vs.* GH-untreated TS patients, respectively, P= 0.639). GH-treated TS patients started GH therapy at a chronological age (CA) of 11.2 ± 3.7 yr, bone age of 9.3 ± 3.1 yr, height SDS (British 1965 standards) -3.1 ± 1.1. GH mean dose was 48μg/kg.d and GH treatment duration was 5.4 ± 3.0 yr. Estrogen replacement was started late, at CA of 14.3 ± 2.0 yr in GH-treated and at 14.9 ± 1.9 yr in GH-untreated patients, and the rate of spontaneous puberty was similar between the groups (GH-treated 16.8% *vs.* GH-untreated 22,8%, P=0.304). Adult height was significantly higher after GH treatment (150.1 ± 5.8 cm *vs.* 143.3 ± 7.2 cm in GH-treated *vs.* untreated TS patients, respectively, P < 0.001), even with a small but significant difference in target height between the groups (158.2 ± 4.8 *vs.*