The MN and MD ΔCV for PTs with a SPC was 10.68% ±2.65% and 11.09% RSP, with a RG of -100.00% to 763.94%. The MN and MD ΔPOGO by the cyst for PTs with a SPC was 78.33% ±369.96% and -31.34% RSP, with a RG of -100.00% to 1734.79%.
The MN and MD ΔCV for PTs with a LPC was -24.60% ±51.89% and -26.57% RSP, with a RG of -88.57% to 91.38%. The MN and MD ΔPOGO by the cyst for PTs with a LPC was -22.79% ±44.90% and -40.46% RSP, with a RG of -80.95% to 47.11%.
Statistical analysis showed no significant %ΔCV or %ΔPOGO when comparing male vs. female, SPC vs. LPC, GHD vs. ISS, or pre-pubertal vs. pubertal PTs. Analysis of ΔPOGO of the 24 SPC PTs demonstrated that 4 (17%) of them developed into LPCs. Analysis of the 10 LPC PTs showed that 6 (60%) of them shrunk into SPCs, one of which re-enlarged into a LPC, and another of which fluctuated between LPC and SPC over a period of 7.34 yrs and 9 sequential MRIs. None of the PTs experienced significant sequelae related to their PCs.
Conclusion: CV can change greatly over time, however few sequelae should be expected. LPCs tend to demonstrate major changes in size and should be tracked for CV change. A minority of SPCs will develop into LPCs. Prediction of change in CV over time requires more sequential data. Change in CV did not appear to be influenced by GH therapy.

Pediatric Endocrinology

PEDIATRIC OBESITY, THYROID, AND CANCER

Various Subcutaneous Continuous Glucose Monitors Comparably Lower HbA1c in Children

Steven Gold, BS[1], Liam McGuirk, n/a[1], James Haigney, MD[2], Jane Torres, MS[1], Tara Patale, n/a[1], Alice Alexandrov, BS[1], Zeyad El-Naghy, n/a[1], Nicholas Andrew Krasnow, n/a[1], Tavia Buysse, BS[1], Richard A. Noto, MD[1].
[1]New York Medical College, Valhalla, NY, USA, [2]Beth Israel Deaconess Medical Center, Boston, MA, USA.

MON-086
Background: Preliminary studies have demonstrated improvement in metabolic control of patients (PTs) using subcutaneous Continuous Glucose Monitoring systems (CGMs). In this study, we investigated the effect of CGMs on PTs' glycemic control and compared the change in patient HbA1c levels between sensors.
Objective: To determine how CGMs affect metabolic control in PTs and the effect of different sensors on glycemic control.
Patients and Methods: 33 PTs with Type 1 diabetes mellitus (DM) who began using a CGM between 2017 and 2019 were selected for inclusion. CGM systems used included DexcomG6™, DexcomG5™, DexcomG4™, Enlite™, Guardian 3™, or Medtronic Sure-T™ sensors. **Results:** The mean (MN) age of PTs at initial visit was 15.3 ± 5.1 yrs and the MN age at second visit was 15.8 ± 5.1 yrs. The MN time between visits was 5.0 ± 2.4 months (mos). 6 PTs had follow up (F/U) times less than 3 mos, 18 PTs had F/U times between 3 and 6 mos, 6 PTs had F/U times between 6 and 9 mos, and 3 PTs had F/U times greater than 9 mos. The MN and median (MD) HbA1c at the initial visit for all PTs was 8.28% ± 1.48 and 8.10%, respectively. The MN and MD HbA1c at final F/U for all PTs was 7.57% ± 1.11 and 7.50%, respectively. The difference in MN HbA1c was significant (p<0.001).
The MN and MD HbA1c at the initial visit for PTs with a F/U time less than 3 mos was 7.55% ± 0.77 and 7.75%, respectively. The MN and MD HbA1c at F/U for these PTs was 7.20% ± 0.79 and 7.20%, respectively. The difference in MN HbA1c was significant (p<0.05).
The MN and MD HbA1c at the initial visit for all PTs with a F/U time greater than 3 mos was 8.44% ± 1.53 and 8.10%, respectively. The MN and MD HbA1c at F/U for these PTs was 7.66% ± 1.15 and 7.50%, respectively. The difference in MN HbA1c was significant (p<0.001).
The MN change of HbA1c between visits was not significant between PTs who had 3–6 mo, 6–9 mo, and 9+ mo F/U times (p=0.96)
15 PTs had HbA1c levels less than or equal to 8.0%. The MN and MD HbA1c at initial visit for these PTs was 7.20% ± 0.41 and 7.30%, respectively. The MN and MD HbA1c at F/U for these PTs was 6.75% ± 0.47 and 6.80%, respectively. The difference in MN HbA1c was significant (p<0.001).
20 PTs had HbA1c levels greater than 8.0% at initial visit. The MN and MD HbA1c at the initial visit for these PTs was 9.18% ± 1.47 and 8.80%, respectively. The MN and MD HbA1c at F/U for these PTs was 8.26% ± 1.03 and 8.00%, respectively. The difference in MN HbA1c was significant (p<0.001). The MN change in HbA1c between the high HbA1c group (-.92% ± 1.02) and low HbA1c group (-0.45% ± 0.32) was not significant (p>0.05).
25 PTs used a Dexcom™ sensor while 8 PTs used a Medtronic™ sensor. The MN change in HbA1c was not significant between these brands (p>0.05).
Conclusion: CGMs improve metabolic control in pediatric PTs with Type 1 DM regardless of initial HbA1c. Further, this improved control is sustained over time. Sensor brands appear to be equally effective at achieving this goal.

Pediatric Endocrinology

PEDIATRIC ENDOCRINE CASE REPORTS II

MODY Diagnosis: Missed Opportunity to Avert Insulin Therapy. A Case Series.

Remberto Paulo, MD[1], Salman Kirmani, MD[2], Kristal Anne Matlock, MD[1], Deborah A. Bowlby, MD[1], Terry Headley, -[1].
[1]Med Univ of South Carolina, Charleston, SC, USA, [2]Aga Kahn University, Karachi, Pakistan.

MON-057
BACKGROUND Prevalence of MODY is 1.2% in pediatric diabetes population. SEARCH study reported most of these patients were misdiagnosed as T1DM or T2DM up to 36% and 51% respectively (1). GCK (MODY 2) and HNF1A/HNF4A (MODY3) are the most common forms of MODY. Despite improvement in testing strategy (panel testing instead of step-wise approach) and cost, MODY testing remains underutilized. These conditions do not require insulin therapy, and MODY 2 patients may even be discharged from clinic after diagnosis. We present 4 cases and valuable lessons learned.

MODY 2 Case 1. 4 yo M referred for hyperglycemia in the 300s during surgery. A1c 6.4%. FBG at home 150s, asymptomatic. MGM and MGM's siblings have diabetes. Diabetes autoantibodies (DAA) negative. C-peptide 5.4 (NL 0.78 - 5.19 ng/ml). MODY panel (GeneDx) showed heterozygous mutation in *GCK* gene (c.70 C>T). Patient remains off insulin, family reassured and advised to undergo genetic testing.
MODY 2 Case 2. 8 yo M diagnosed at local ED with "T1DM" after presenting with polyuria, polydipsia, and random BG 237. A1c 6.7%, C-peptide 1.9, started on basal-bolus insulin. MODY panel (sent a year later when patient was found to have low insulin requirement, negative DAA) showed pathogenic variant in *GCK* gene. Weaned from insulin, A1c unchanged (6.3–7%). Mother found to have same mutation. **MODY 3 Case 1.** 16 yo F referred by PCP who started her on insulin a year prior after an incidental finding of hyperglycemia. A1c was 7.5% at diagnosis. Mom, MGM have diabetes, unknown type (MGM thin by report). DAA neg, C-Peptide 1.74. MODY Panel showed *HNF1A* heterozygous gene mutation for RI31Q. She was switched to Glyburide, blood glucose 90s. **MODY 3 Case 2.** 10 yo M referred from the ED for "T1DM" (weight loss, fatigue, A1c 7.6%) started on basal-bolus insulin, but lost to follow up for a year. Brother has MODY 3. DAA neg, C-Peptide 3.1. Targeted gene sequencing showed *HNF1A* gene mutation. He was switched to Glyburide, A1c improved to 6.7%. However, patient became noncompliant as teenager, A1c now 9.3%.
CONCLUSION MODY remains underdiagnosed. A high index of suspicion should be maintained in nonobese, DAA-negative patients diagnosed with DM before 25yo. Although DAA and genetic testing can be costly, diagnosis can dramatically alter diabetes management as illustrated in all 4 cases, and overall cost of management may be lower in the end. Patients with MODY 2 do not develop vascular complications associated with diabetes, nor require pharmacotherapy. MODY 3 patients may be safely switched to sulfonylurea monotherapy, though degree of diabetes control depends on compliance with medication. Testing gives relatives previously misdiagnosed the opportunity to improve their own quality of life. More education for health care providers is warranted for prompt diagnosis and appropriate management of this condition.
Reference:1. Pihoker, et al. JCEM 2013; 98:4055–62

Tumor Biology

ENDOCRINE NEOPLASIA CASE REPORTS I

Lactic Acidosis as a Rare and Unusual Presentation of Pheochromocytoma

Nazar Mohammad, MD[1], Margaret Frith, MD[1], Olayinka Olawale Wilhelm, MD[2].
[1]United Health Services, Johnson City, NY, USA, [2]Wilson Memorial Regional Med Ctr, Vestal, NY, USA.

SUN-933

Background: Pheochromocytoma is a rare catecholamine-producing tumor of chromaffin cells in the adrenal medulla or of a paraganglion. Typically it presents with sustained or paroxysmal hypertension, severe headaches, palpitations and sweating due to hormone excess. However, the presentation can be variable and can mimic many other diseases. If left undiagnosed or untreated, it can lead to life-threatening consequences.
Case Presentation:A 35 year old female with significant past medical history of migraine headaches, poorly controlled hypertension and a recent new onset seizure, presented with progressive worsening shortness of breath and persistent abdominal pain following a gastrointestinal illness. She also reported diaphoresis, cold fingers and toes, abnormal weight gain, and orthostatic symptoms that gradually worsened for two months prior to presentation. Laboratory evaluation revealed lactic acidosis, leukocytosis, and hypokalemia. Subsequently, a CT scan of the abdomen was performed that revealed an adrenal mass with significant elevation in urine metanephrines. As a result, the patient was diagnosed with pheochromocytoma and successfully treated with laparoscopic left adrenalectomy.
Conclusion: Pheochromocytoma is a rare but can be life threatening if left undiagnosed. It is of utmost importance for clinicians to keep in mind such unusual presentation of a potentially life threatening tumor. To the best of our knowledge, this is an unusual presentation of Pheochromocytoma with severe lactic acidosis.

Thyroid

THYROID CANCER CASE REPORTS I

Mass Cord Compression: Metastasis of Insular Thyroid Cancer

Dina Jaber, MS[4,1], Bianca Vazques, MD[2], Gary Nagamoto, MD[3], Mohamad Hosam Horani, MD[4].
[1]Midwestern University, Gilbert, AZ, USA, [2]AZ Thyroid surgery, Gilbert, AZ, USA, [3]CRMC, Chandler, AZ, USA, [4]Alsham Endocrinology, Gilbert, AZ, USA.

SUN-490

Intro: Insular thyroid cancer is a rare and complex form of thyroid cancer, often referred to as poorly differentiated carcinoma. The exact incidence of insular thyroid cancers is difficult to assess due to controversial classification of this thyroid cancer over the years. It is termed poorly differentiated as it falls between the well-differentiated and undifferentiated carcinomas both morphologically and biologically[1].
Case: A 41 year old Hispanic female, with a history of prolactinoma and hyperparathyroidism, presented to the hospital with 10 days of progressive lower extremity weakness and paresthesias from T4 downwards, inability to bear weight, and no bowel movement for 12 days. MRI revealed a large thoracic soft tissue mass (7x4x4cm) centered in the posterior and medial aspect of the chest wall at T4-T5 with involvement of the spinal cord and vertebral bodies. She was also found to have a right sided thyroid mass (4.5x5x4 cm) with tracheal deviation-
HerThyoid function test, were normal Intact PTH was 261, Thyroglobulin over 300, and Thyroid Antibodies were negative. Patient underwent T3-T6 laminectomy, T2-T7 fusion, and T4-T5 tumor resection, which was subtotal due to vascularity. Second procedure included a right thoracotomy, chest wall resection of ribs 4 and 5 with full resection of paraspinal mass, total thyroidectomy, parathyroidectomy