clitoral hood, labia majora, hypogastrium and upper legs. In our case the baby presented with vaginal bleeding at 45 weeks that recurred about 3 weeks later and resolved by 50 weeks, with only minimal swelling. This case underlines the need to be aware of this etiology and its varied presentation. [1] Preterm ovarian hyperstimulation syndrome presented with vaginal bleeding: a case report. Altuntas et al. J Pediatr Endocr Met; 273(3-4):355-358.

Pediatric Endocrinology

PEDIATRIC ENDOCRINOLOGY CASE REPORT

A Case of Psuedohypoparathyroidism Unmasked by COVID-19 and Rhabdomyolysis

jacqueline chan, MD, FAAP, Asif Mansuri, MD.
Medical College of Georgia at Augusta University, Augusta, GA, USA.

Background: Psuedohypoparathyroidism type 1 b (PHP 1b) is a rare condition characterized by hormone resistance with PTH. It is caused by imprinting defect of the GNAS gene and is acquired as autosomal dominant. Compared with Pseudohypoparathyroidism type 1a (PHP1a), PHP1b does not have the characteristic physical features know as Albright hereditary osteodystrophy or AHO. **Results:** 12-year-old female with unremarkable past medical history presented with seizures. She has been complaining of leg pain for a week but on the day of presentation noted to have stiffening and shortness of breath. There was no history of cough, fever, leg trauma, headache, dysuria, hematuria or dark urine. She had no significant family history. Work up revealed severe hypocalcemia of 4.6 mg/ dL and elevated phosphorus (7.1mg/dl). There was no hypoglycemia and other electrolytes including renal function were normal. Patient also had normal inflammatory markers, normal fibrinogen, and normal ferritin. Urine was positive for trace protein and was positive for myoglobin. Alkaline phosphatase was normal. Urine toxicology screen was negative. Hand X-rays did not showed the shortening of metacarpal bones and kidney ultrasound was normal. After IV calcium bolus, calcium barely increased at 5.0. Interestingly CK was noted to be elevated at 3,794 U/L. Patient physical exam was normal and there was no signs of Albright hereditary osteodystrophy (AHO). Patient was positive for COVID19. Patient required intensive fluid therapy to correct CK which increased up to 11,223 U/L on 3rd day of admission. It eventually came down back to normal on the 6th day. Creatinine levels remained normal. Patient continued to receive high dose calcium and calcitriol supplement and discharged with calcium of 8.5 mg/dL and phosphorus of 7.8 mg/dL. Additional work up showed PTH of 885 pg/mL consistent with pseudohypoparathyroidism. Vitamin D levels and thyroid function were normal. Genetic testing for pseudohypoparathyroidism is awaited. **Conclusion:** Psuedohypoparathyroidism type 1 b (PHP 1b) is a rare endocrine disorder presenting with hypocalcemia, hyperphosphatemia and increased PTH values due to a variable resistance to target organs. As in our case it was unmasked by COVID 19 infection and rhabdomyolysis. Best of our knowledge there is no such unusual case reported in children. Severe hypocalcemia likely due to combination of factors including tissue calcium deposition, hyperphosphatemia, and skeletal resistance to PTH. As published in some recent reports, this case also illustrates that rhabdomyolysis could be potential complication of SARS- CoV2 infection in early stage with normal renal function, which warrants further research.

Pediatric Endocrinology

PEDIATRIC ENDOCRINOLOGY CASE REPORT

A Case of Severe MIS-C in Pediatric Diabetes: Complications of COVID-19

Alyssa M. Dye, MD, Sandra R. Arnold, MD, Terri H. Finkel, MD, PhD, Ahlee Kim, MD.
University of Tennessee Health Science Center, Memphis, TN, USA.

Background: Multisystem inflammatory syndrome in children (MIS-C) is a serious inflammatory response to a prior coronavirus disease of 2019 (COVID-19), characterized by fever, inflammation, and multiorgan dysfunction. Current literature does not indicate a relationship between pediatric diabetes and risk of developing MIS-C. Here, we report a case of pediatric type 2 diabetes (T2D) with diabetic ketoacidosis (DKA) and severe multi-organ dysfunction with SARS-CoV-2 serology positivity.
Clinical Case: A 13-year-old African American female with obesity (Body Mass Index >99th %) and poorly controlled T2D (HbA1c 12.8%) presented to the emergency department for one week of sore throat, headache, and abdominal pain. SARS-CoV-2 positivity was confirmed by PCR. The next day, she was found unconscious at home. She was diagnosed with DKA and was directly admitted to the pediatric intensive care unit. IV insulin and aggressive fluid resuscitation were initiated and her DKA resolved over the next 72 hrs. However, she exhibited continued altered mental status with slurred speech and significant respiratory distress requiring respiratory support. Due to the severe presentation and multi-organ involvement, a multi-disciplinary care team was formed. Further workup confirmed acute respiratory distress syndrome with pneumonia; severe acute kidney injury (AKI, creatinine of 4.56 mg/dL); presumed myocarditis (ST elevation on EKG, troponin 4.47 ng/mL, BNP 129.8 pg/mL); punctuate intraparenchymal hemorrhage in the splenium of the corpus callosum; transaminitis (AST 188 u/L, ALT 100 u/L); pancreatitis (amylase 651 u/L, lipase >9500 u/L); thrombocytopenia with consumptive coagulopathy (platelet 81 X 10^3/μL, d-dimer 5.91 mcg FEU/mL), increased inflammatory markers (ESR 53 mm/hr, ferritin 127 ng/mL), and positive SARS-CoV-2 serology. A presumed diagnosis of MIS-C was made per the Centers for Disease Control and Prevention definition and she was started on dexamethasone and intravenous immunoglobulin (IVIG). While consideration was given to the possibility of acute COVID-19 infection in combination with DKA, she was not a candidate for remdesivir due to AKI. On day 12, she developed new dysarthria, dyspraxia and behavioral changes. Encephalopathy workup was negative (CSF Encephalopathy autoimmune panel negative, NMDA receptor negative) and she was restarted on dexamethasone and IVIG. She was discharged on day 28.
Conclusion: There is a paucity of literature of MIS-C associated with COVID-19 in the pediatric diabetes. Our case

highlights several novel aspects of MIS-C with concurrent poorly controlled diabetes and DKA, including severe central nervous system manifestations and prolonged hospitalization. Further studies are warranted to elucidate an association between pediatric diabetes and MIS-C and to develop guidelines for management of MIS-C in poorly controlled pediatric diabetes.

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PEDIATRIC ENDOCRINOLOGY CASE REPORT

A Case Series of Four Preterm Intrauterine Growth Restricted Babies With Transient Hyper-Insulinemic Hypoglycemia and Cholestasis

Pratibha Rana, MD[1], Megan Rodrigues, Med student[2], Chaitali N. Mahajan, MD[1], Michael F. Nyp, DO[1], Pandey Vishal, MD[1].

[1]Childrens Mercy Hospital, Kansas City, MO, USA, [2]University of Kansas, Kansas City, KS, USA.

Background: Premature infants with intrauterine growth restriction (IUGR) are predisposed to stress related hyper Insulinemic hypoglycemia (HIH). These babies are at risk for other prematurity related complications including direct hyperbilirubinemia. However, association of HIH with this has not been described, and transient cholestasis in HIH infants has not been reported. We present 4 such infants with perinatal stress related HIH who had cholestasis that resolved with time. **Case series:** In our retrospective review of these preemies with IUGR who had developed HIH, we found that 4 infants developed direct hyperbilirubinemia. Their gestational ages at birth ranged between 26 to 27 weeks, with birth weights between 527 to 642 grams. These infants had received total parenteral nutrition (TPN) for durations ranging between 12 to 19 days of life (DOL). HIH was established in them at variable ages between 55 to 75 DOL, based on an exaggerated glycemic response to glucagon. Of these, 1 baby was not started on Diazoxide due to underlying fluid overload. His HIH resolved by DOL 182. Two babies responded to therapy and while one remained on this till its resolution at 9 months age, another had the Diazoxide discontinued due to acute respiratory worsening leading to readmission. HIH in the latter resolved by 109 DOL. Fasting The last baby developed fluid overload early in therapy leading to its discontinuation without establishing response. Hypoglycemia in these infants resolved by ages between 4 to 9 months of life. Interestingly direct hyperbilirubinemia was noted by age 16 to 59 DOL. In all infants, the diagnosis of HIH was established after the onset of cholestasis. Extensive work up for hyperbilirubinemia ruled out any organic pathology. This transient cholestasis was noted to have resolved by ages 80 to 115 DOL. **Conclusion:** It appears from our experience in these premature infants, cholestasis may be associated with HIH. Its diagnosis preceded the establishment of HIH. We noted that HIH diagnosis was delayed by around 30 days after the onset of intermittent hypoglycemia. Both the cholestasis and HIH were transient. Whether the cholestasis may prognosticate the development of HIH or is indicative of transient HIH needs to be investigated. Any association between the two needs to be studied to address a common causality. IUGR babies with conjugated hyperbilirubinemia develop a mild and transient HI state which is self-resolving. Due to transient nature of this HIH in these IUGR babies with cholestasis, a genetic work up for HIH may be deferred.

Pediatric Endocrinology

PEDIATRIC ENDOCRINOLOGY CASE REPORT

A Novel Case of Hyperinsulinemic Hypoglycemia in a Neonate With SARS-CoV-2 Infection

Remberto Paulo, MD, Deborah A. Bowlby, MD, Kristal Anne Matlock, MD.

Medical University of South Carolina, Charleston, SC, USA.

The Case: An 8 days old male, born at 40 wks, 2.8 kg (SGA) presented to local ED with lethargy, decreased PO intake and urine output, respiratory distress. No fever, URI sx, vomiting/diarrhea. His dad had URI symptoms and fever a week prior. He was found to have T=95.5F, glucose <10 mg/dL, improved to 125 after D10 boluses x2; and required supplemental O2 due to desats/cyanosis. CXR showed bilateral hazy opacities. Sepsis rule out was initiated, patient admitted to the PICU, started on antibiotics and dextrose. Patient became more alert over the next 3 days, but could not be weaned off from IV dextrose/continuous feeds, GIR up to 15 mg/kg/min. He was transferred to our institution. Critical sample at BG of 45 mg/dl showed high insulin level (6.9 uU/ml) and C-peptide (1.25 ng/dl); low beta-OH butyrate <0.2 mmol/L; and free fatty acids (0.25 mmol/L); all suggestive of hyerperinsulinemic hypoglycemia (HH). Cortisol and GH robust at 10.5 mcg/dl and 7.13 ng/ml. Nasopharyngeal swab for SARS-CoV-2 RT-PCR positive. Dad's swab also positive. Mom was asymptomatic and not tested. ID was consulted, recommended supportive management and close observation. Pt was started on Diazoxide 10mg/kg/day divided q8h, and hydrochlorothiazide (HCT) 5mg/day. Patient's status gradually improved - BG stabilized, feeds were compressed, IV fluids and O2 supplementation weaned off, and was discharged after 8 days with average BG in the 70-80's range. Diazoxide and HCT were successfully weaned off in the following 3 mos. To this day patient remains well, no recurrence of hypoglycemia.
Discussion/Conclusion: There is a dearth of information on SARS-CoV-2 infection in newborns. The few studies available show favorable outcomes in this population, with typical mild-moderate respiratory symptoms and fever, while some newborns are asymptomatic. Our patient required oxygen tx and developed HH requiring Diazoxide therapy. To our knowledge, this is the first reported case of HH in the newborn with SARS-CoV-2 infection. Hyerperinsulinism is the most common cause of hypoglycemia in infants. These newborns are at risk of developing significant neurologic morbidity, which can be dose dependent. Prompt diagnosis and aggressive management are important to reduce such risk. Perinatal stress is likely the underlying mechanism leading to HH in newborns with SARS-CoV-2 infection. Patient is also SGA. Both perinatal stress and SGA can lead to inappropriately elevated insulin levels and resultant hypoglycemia. HH in both of these conditions is effectively managed by Diazoxide. Our case illustrates that although most newborns do well with SARS-CoV-2 infection, a high index of suspicion for HH should be maintained in such