5-year-old female with hypoglycemia
Endorama January 19, 2012 Rochelle Naylor, MD

Chief complaint and HPI
- 5-year-old female transferred from OSH to PICU with lethargy, dehydration, and low blood sugar
- USOH until 2 days PTA
- N/V, no po intake except small amounts of water
- To OSH ER day PTA- dx'd w/ UTI, d/c'd home w/ Abx
- DOA cont’d n/v, lethargic → taken to ED

HPI, continued
- Exam consistent with moderate-severe dehydration
- Cont’d lethargy after total 40 ml/kg IVF bolus
- Labs significant for serum BG of 52
- Rev’d D10 (2 ml/kg), placed on D5NS at 1500 ml/m2/d prior to txfer to Comer ICU

OSH labs
- Head CT- negative
- Urine toxicology screen- negative

Hospital Course
- Continued on IVF in PICU w/ improvement
- Tolerated small po intake x I
- DOA= emesis of water and lethargy
- POC BG= 44
- Serum BG= 45
- IVFs of D5NS resumed, Peds Endo consulted for hypoglycemia

Past Medical History
- FT, NSVD, no complications
- Autism spectrum disorder
- GERD
- UTIs x3
- Pneumonia x2
- Previous episodes of lethargy
  - During illnesses w/ poor po intake
  - 2 yr old after refusal to eat in the care of her GP a ~16 hours
- Medications- None
- Allergies: Azithromycin- delerium

FAMILY HISTORY
- Negative for DM
- Negative for hypoglycemia

SOCIAL HISTORY
- LAHW parents

REVIEW OF SYSTEMS
- Nausea, vomiting- now resolved
- Appetite improved, tolerating po intake
- No concern for ingestions
- No access to hypoglycemic agents

PHYSICAL EXAMINATION
- Wt: 20 kg (75th); Ht: 102.8 cm (15th)
- Gen: WD, WN; NAD
- HEENT: W/o dysmorphic features; PERRL; MMM; NL thyroid examination; neck supple
- CV: RR, NL, S1, S2; no murmurs
- Puls: CTA b/v
- Abd: Soft, NT, NO, no masses
- GU: Tanner I
- MSK: NL ROM, No edema, TD, deformity
- Neuro: Nonfocal; 2+ DTRs

DIFFERENTIAL DIAGNOSIS FOR HYPOGLYCEMIA IN A CHILD?
**DDx of hypoglycemia in children**

- **Ketones absent**
  - Hyperinsulinemia
  - Congenital
  - Beta-cell adenoma
  - Rebound hypoglycemia
  - Medication
  - Munchhausen by proxy
  - Fatty acid, organic acid oxidation defects

- **Ketones present**
  - Idiopathic Ketotic hypoglycemia
  - IEMs
  - GH deficiency
  - Cortisol deficiency
  - Ingestions (alcohol, salicylates)

**EVALUATION?**

OSH labs

- Anion gap: 24
- Day PTA Urine ketones: 40, glc: neg
- DOA Urine ketones: 150, glc: neg

**Laboratory evaluation**

- Beta-hydroxybutyrate: 6.84
- Lactic acid: 1.1 (later that day w/ BG-146, bicarb 20)
- Cortisol: 43.2
- GH: could not be added
- HbA1c: 5.1
- POC BGs: 91-154 throughout the remainder of the hospitalization

**Idiopathic ketotic hypoglycemia**

- Most common form of hypoglycemia in young children
- Hypoglycemia and ketosis uniformly provoked by a brief fast after hypocaloric high fat, low carbohydrate diet
- Characteristic inability to respond to glucagon after brief fast
- Usually appears between the first and third years of life, and remits spontaneously by age 6-8

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- Alanine levels significantly lower at the end of the test in hypoglycemic subjects
- Ketones, FFA levels were WNL during duration of fasting in all subjects

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Mechanism of IKH—Pagliara, et al

Objective: determine if the primary defect in IKH is a deficiency of gluconeogenic precursors or an abnormality in the hepatic gluconeogenic enzyme system

- 8 children with IKH and 7 age-matched controls

Conclusion: A deficiency in gluconeogenic precursor (alanine) rather than a defect in the hepatic gluconeogenic enzyme apparatus is the most likely etiology of IKH

Utility of GH and cortisol values during hypoglycemia

- Study aim: To determine if GH and cortisol obtained during fasting hypoglycemia can identify children with deficiencies

- Retrospective chart review of all diagnostic fasting tests (n=151)

- Hypoglycemia defined as SBG ≤ 50 mg/dL

- NL GH level defined as ≥ 7.5 ng/mL

- NL cortisol level defined as ≥ 18 mcg/dL

- 70% had GH and cortisol levels below “normal” thresholds
Summary

- Idiopathic ketotic hypoglycemia is the most common form of hypoglycemia in young children
- IKH is caused by the inability to sustain adequate glucose production related to decreased alanine supply
- While GH and cortisol deficiency need to be ruled out, the utility of assessment at the time of hypoglycemia is questionable

References

- CJ Elder, VJ Wright, NP Wright. Time to end the routine testing of growth hormone and cortisol on hypoglycemia screens? Arch Dis Child. 2009; 94(12):1000-1. DON'T HAVE ARTICLE