03/11/24 Morning Report with @CPSolvers

*"One life, so many dreams" **Case Presenter**: Robert Weber (@RobWeberMDPhD) **Case Discussants**: Maddy (MadellenaC@) and Kirtan (KirtanPatolia@)*

CC: Code stroke

HPI:
85yo F with DM, HTN, hypothyroidism brought in by EMS as a code stroke

Woke up at 6am, found by family to be slurring words, unable to feed herself breakfast.
No falls, no head trauma

In the field, finger stick glucose was 73. She was given IV dextrose and brought to the hospital. By the time she was in the hospital, symptoms had resolved.

In the ED, CT head and neck were negative. She had recurrence of symptoms. POC and serum glucose was 55 and was started on d10 with improvement in her glucose. With these episodes, no sweating, no dizziness, no tremors.

Has had episodes of sleepwalking w/ disorientation, amnesia over the last year. Has had ongoing lows in her sugars at home with snacking, particularly at night. Has had unintentional weight loss, down to 110 lbs over the last year.

PMH:
Diabetes, last HbA1c: wnl.
Hypothyroid
HTN

Meds:
Losartan 50mg
Levothyroxine 50 mcg

Fam Hx:
N/A

Soc Hx:
Previously independent, in the last year now lives with family members

Health-Related Behaviors:
No alcohol/substance use

Allergies:
N/A

Vitals: T: 37C **HR:** 76 **BP:** 150/56 **RR:** wnl **O2 sat**: 94% on 2L
Exam:
Gen: Thin, cachetic
HEENT: Temporal wasting. No gum hyperpigmentation. No goiter
CV: wnl.
Pulm: Markedly diminished breath sounds on right, and base.
Abd: Flat, non-obese. No striae.
Neuro: wnl.
Extremities/skin: Scalloped clavicles, redundant skin in upper arms. Marked decreased muscle mass in the thighs. No rash, bruise.

Notable Labs & Imaging:
Hematology:
WBC: 6 Hgb: 11 Plt: 120

Chemistry:
Na: wnl K: wnl Cl: wnl HCO3: wnl BUN: wnl
AST: 15; ALT: 14; Alk-P: 69; T.bili 0.4
Cr: 89 CR: 0.62. Cystatin-C: 1.91 w/ eGFR 27
VBG: 7.42. Lactate 1.0

TSH 3.6.
Cortisol 12 with ACTH stim → 28 (>18: sufficient).
A1c 4.9%
Glucose 63
C-peptide: 0.2 (nml 0.8-3.5)
Insulin: undetectably low
Proinsulin: undetectably low
Sulfonylurea screen: negative
Beta hydroxybutyrate: 0.25 (nml 0-0.28)
Insulin receptor antibody: Neg.
IGF-2: 50 Paired IGF-1: 37 (IGF-2 mediated if IGF-2:IGF-1 > 10)

Imaging:
CXR: Large R-sided pleural effusion with a 3x3 cm mass within the effusion.
Head CT w/o contrast: Negative for acute intracranial abnormalities.
CT AP w/ contrast: pancreas unremarkable.
Tumor bx: stromal tumor consistent with a solitary fibrous tumor.
Dx: Hypoglycemia due to renal insufficiency, poor PO intake, cachexia.

Problem Representation:
85 y/oF with DM, HTN, hypothyroidism brought in as a code stroke, found to have renal insufficiency, cachexia, and hypoglycemia improved with glucose.

Teaching Points (Tansu):
Hypoglycemia: Whipple's triad: symptoms, lab proof, resolution of symptoms w/ tx.
Approach: Hypoglycemia → Real vs. not real → Real → Insulin mediated vs. Not. → Insulin-independent → (1) Decreased intake; (2) Decreased gluconeogenesis (liver, kidney dz); (3) Increased consumption (Warburg effect); (4) Medications (insulin, C-peptide, sulfonylurea levels; other people in the household using meds?). // Finger stick has issues (**Type 1** pseudohypoglycemia): Tiny capillaries, false lows. Gold standard: venous (though **Type 2** pseudohypoglycemia w/ leukocytosis, PV, paraproteinemia also possible). // Symptoms only at night: nocturnal hypoglycemia? pathology in CNS worsened by hypoglycemia? // T1DM: pts are more prone to wt loss vs. T2DM.// Hypoglycemia + losing weight → **Catabolic state?**
Non-islet cell hypoglycemia (NICH): From excessive production of IGF-2 and its precursors → stimulates insulin receptor (seen in large mesenchymal and epithelial tm; dx by: IGF-2/IGF-1 ratio). // She may not have neuroglycopenic symptoms of hypoglycemia, because that response is dampened w/ repeated episodes. // Chronicity in physical exam w/ scalloped clavicles, temporal wasting. // Tumoral overproduction of IGF2 stimulates insulin receptors → hypoglycemia.// Paraneoplastic Abs → hypo/hyperglycemia (can bind insulin, or insulin receptor).// Presence of insulin → Ketones.// Kidney: ⅓ of gluconeogenesis; renal toxin build up inhibits liver gluconeogenesis. Cachexia → No mobilization of stores.