enzymes normalized when her glucose levels normalized and DKA resolved.

Further work-up ruled out more common etiologies of liver injury. Multiple abdominal ultrasounds and CT scans showed a normal sized liver without obvious structural abnormalities. Labs were significant for negative hepatitis B and hepatitis C; several negative anti-smooth muscle, anti-nuclear antibody, centromere antibody, and liver kidney microsomal type 1 antibody; normal levels of ceruloplasmin and alpha 1 anti-trypsin; low iron levels 23 ug/dL (60-180 ug/dL); borderline low IgG 627 mg/dL (700-1600 mg/dL).

We hypothesized that the patient likely had GH by exclusion of other liver pathologies and given the context of transient transaminitis during DKA.

**Conclusion**: GH is a benign and favorable diagnosis in diabetic patients with elevated transaminases.[1] Given the small number of cases of GH reported, there is a need to record and analyze more patients with likely GH in order to better understand the condition. Appropriate clinician awareness of GH can also eliminate the need for time consuming and costly workup.

**References**:1. Sherigar, Jagannath M et al. "Glycogenic Hepatopathy: A Narrative Review". World Journal Of Hepatology, vol 10, no. 2, 2018, pp. 172-185. Baishideng Publishing Group Inc., doi:10.4254/wjh.v10.i2.172.

# Diabetes Mellitus and Glucose Metabolism

## METABOLIC INTERACTIONS IN DIABETES

### *Novel Elisa Assays Demonstrate Specificity of Islet and Intestinal Processing of Proglucagon.*

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**SUN-LB124**

Circulating proglucagon peptides (PGP) are produced in islet α-cells, enteroendocrine L-cells. Release of PGP is thought to be tissue specific, e.g. α-cells make glucagon and L-cells make GLP-1 through predominant actions of proconvertases 2 and 1/3 (PC2 and PC1/3). However, this dichotomous model has recently been challenged. To address the contribution of the gut and pancreas to plasma PGP we developed 4 novel sandwich ELISA assays and applied them in studies with PGP stimulation from the islet (IV arginine) and intestine (meal). Monoclonal antibodies were raised in mice with genetic ablation of proglucagon transcription. Clones were screened and selected for affinity and specificity, and assays for glucagon, GLP-1, glicentin and oxyntomodulin developed. Eight healthy humans received 5 g arginine intravenously after a 12 hour fast and had blood sampled for 15 minutes; an additional 10 consumed a liquid mixed nutrient meal and prandial blood was taken for 180 minutes. None of the assays registered signal in plasma from proglucagon null mice, and specificity, background and cross-reactivity were acceptable in each. In response to IV arginine plasma glucagon increased 4-fold, and GLP-1 1.5-fold, with significant increases in 15-minute AUC; there was no significant change in either glicentin or oxyntomodulin. In response to meal ingestion there was no change in circulating glucagon, but oxyntomodulin, GLP-1 and glicentin increased 2, 3, and 4-fold respectively. These findings are generally compatible with PC1/3 dominant processing of PGP in the gut, but raise the possibility that α-cells produce both PC2 (glucagon) and PC1/3 (GLP-1) products.

# Diabetes Mellitus and Glucose Metabolism

## CLINICAL AND TRANSLATIONAL STUDIES IN DIABETES

### *A Phase 2 Evaluation of a Ready-To-Use Liquid Stable Continuous Subcutaneous Glucagon Infusion for the Treatment of Hypoglycemia Associated Autonomic Failure*

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**MON-LB110**

**OBJECTIVE:**A novel, ready-to-use, liquid stable, continuous subcutaneous glucagon infusion (CSGI; Xeris Pharmaceuticals) was evaluated for the treatment of adults with type 1 diabetes (T1D) with documented Hypoglycemia Associated Autonomic Failure (HAAF).

**METHOD:**This was a Phase 2 prospective, multi-center, randomized, placebo-controlled, double-blind, parallel trial in T1D adults with documented HAAF, defined as Gold Scale Score ≥4. Subjects were randomized in a 1:1:1 ratio to receive 4 weeks of continuous treatment (via Omnipod pump) with high rate CSGI (80mcg/hour), low rate CSGI, (20mcg/hour) or placebo (matched infusion rates for low and high rate CSGI). The primary endpoint evaluated at 4 weeks was the percent change of the epinephrine hormone response after 30 minutes of induced hypoglycemia. Epinephrine levels and hypoglycemia symptoms were recorded following a stepwise hypoglycemia clamp, and results compared between study treatment arms. Following the first four weeks, subjects continued for an additional 24 weeks to assess their epinephrine hormone response to hypoglycemia measured at 3 months post-treatment (16 weeks), and 6 months post-treatment (28 weeks).

**RESULTS:**Forty-nine subjects were randomized to receive treatment with high rate CSGI (n=15), low rate CSGI (n=18), or matching placebo (n=16). At the end of study treatment, there were no statistically significant differences between the treatment arms for percent change of epinephrine hormone response during a stepwise hypoglycemia clamp (CSGI vs. placebo; p=0.160). As a result, long-term follow-up of the subjects was stopped early. The long-term follow-up data collected to date will be reported separately. The administration of low and high rate CSGI was associated with mild to moderate nausea (5.9%, 20%,