Introduction: Fasting hypoglycemia in chronically ill patients with kidney disease is frequently multifactorial. Patients with chronic kidney disease are prone to hypoglycemia due to decreased renal clearance of insulin, impaired renal gluconeogenesis and increased prevalence of other medical problems and medications. However, ruling out the presence of an insulinoma can be difficult in this patient population due to the impact of kidney disease on laboratory evaluation of hypoglycemia. **Case:** A 43-year-old male with history of paraplegia from gunshot wound, neurogenic bladder, colostomy, chronic osteomyelitis, frequent polymicrobial bacteremia was noted to have persistent, asymptomatic hypoglycemia during multiple inpatient hospitalizations dating back to 2007. He was taking no medications associated with hypoglycemia. Following normal ACTH stimulation and thyroid testing, he underwent a 72-hour fast in 2017 when his creatinine was 1.32 mg/dL and GFR was >60; BMI was 22.32 kg/m 2 with prealbumin of 15 mg/dL. The fast was concluded after 23.5 hours with serum glucose of 46 mg/dL, insulin level of 3.1 uIU/mL, c-peptide of 2.4 ng/mL, undetectable proinsulin, and beta-hydroxybutyrate of 0.62mmol/L and a negative sulfonylurea screen. Administration of glucagon at time of fast completion improved glucose to 65 mg/dL. He was started on diazoxide 85mg every 8 hours during that hospitalization, which he discontinued without medical recommendation in 2018. He developed end stage renal disease requiring hemodialysis in 2019. During an admission in 2020, serum fasting glucose as low as 30mg/dL. Further laboratory evaluation at that time revealed glucose of 47 mg/dL, with insulin level 39.1 uIU/mL, c-peptide 18.2 ng/mL, and proinsulin of 20.1 pmol/L. Multiple CT abdomen imaging studies confirmed normal pancreas. Diazoxide 110mg every 8 hours was resumed and patient had decreased frequency of hypoglycemia episodes. Diazoxide has since the tapered to 80mg twice daily without increase in hypoglycemia frequency, with goal to taper further in the future. **Conclusion:** Here we present the case of an individual patient who underwent laboratory evaluation of hypoglycemia before and after development of end stage renal disease with profound differences in lab results despite similar degree of hypoglycemia. It has been demonstrated previously that c-peptide and insulin levels are elevated in patients with end stage renal disease and that these labs decrease with hemodialysis while remaining above normal range. The elevation of c-peptide in this population is thought to be due to the kidney being the main site of beta-cell polypeptide degradation. Additionally, this case suggests successful use of diazoxide therapy in patients without classic endogenous hyperinsulinism.

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Insulinoma Evaluation Before and After Development of End Stage Renal Failure: A Case Report

Jillian Pattison, DO and Jennifer Merrill, MD