boluses in the first 96 hours of hospitalization. His glucose infusion was transitioned to D5 and then discontinued. Psychiatry determined the patient was no longer a risk to himself and outpatient therapy was warranted. His blood glucose was well controlled and he was not discharged with insulin.

Discussion: Insulin glargine is a long-acting human insulin analogue with a prolonged activity profile and no pronounced peak. At doses between 0.4 IU/kg to 0.8 IU/kg, insulin glargine is metabolized in 24 hours. However, in cases of insulin overdose, insulin effects and absorption are highly variable. Multiple studies have illustrated the unusual prolonged hypoglycemic effect of glargine after massive doses. The mechanism behind this is still unclear but several factors may result in this prolonged duration of action. Larger injection volumes create a larger depot that may have a mechanical effect on the microcirculation leading to delayed absorption. Presence of lipodystrophy from repeated insulin injection, hepatic impairment, and renal impairment may also alter insulin kinetics. Another contributing factor in our patient is obesity, which is associated with delayed insulin absorption.

Conclusion: Insulin glargine overdose can lead to prolonged hypoglycemic effect due to altered pharmacokinetics. Physicians should be cognizant about this and closely monitor blood glucose levels and anticipate the possibility for prolonged IV dextrose infusion.

Diabetes Mellitus and Glucose Metabolism

DIABETES CASE REPORTS

Pseudohypoglycemia: A Simple Approach to Complex Phenomenon

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Background: Pseudohypoglycemia is a condition where the measured glucose level is falsely lower than the actual level. It can be due to impaired circulation or in vitro glycolysis. **Clinical Case:** Patient 1: A 58-year-old male with a past medical history of sickle cell trait was admitted for alcohol intoxication. Low blood glucose levels were found on finger stick point-of-care testing (POCT). Fasting and postprandial glucose levels were between 17 and 60 mg/dL. The patient was asymptomatic, alert with no tremor, palpitation, or sweating. Glucose POCT was repeated from the earlobe each time a low finger stick level was found, showing normal glucose levels ranging between 85 to 115 mg/dL, confirmed with venous blood glucose measurement. On examination, the patient had mild skin thickening of the hands, with no acrocyanosis, calcinosis, or finger ulceration—normal peripheral pulses with no signs of heart failure. Lab results showed macrocytic anemia, otherwise normal metabolic panel. ANA, anti-Scl-70, and anticentromere antibodies were negative. Patient 2: A 91-year-old female admitted for decompensated heart failure was incidentally found to have fasting glucose levels on finger stick POCT ranging between 30–50 mg/dL. Postprandial glucose on finger stick POCT was ranging between 55–120 mg/dL. Venous glucose levels were ranging between 90 and 180 mg/dL. The patient was alert and asymptomatic. Examination showed bilateral acrocyanosis, poor peripheral circulation with capillary refill > 5 seconds, pitting pedal edema, elevated JVP, and crackles on chest auscultation. Labs showed BNP levels of 1130 pg/mL (n < 100 pg/mL), with ejection fraction of about 35% on echocardiography. Heart failure management was optimized, and warming of the hands with blankets improved peripheral circulation, with capillary refill < 2 seconds, and resolution of cyanosis coinciding with matching of finger stick POCT glucose levels to that of venous blood. Both patients were asymptomatic at the time of low POCT glucose and thus did not fulfill Whipple's triad (measured hypoglycemia, symptomatic, a reversal of symptoms on glucose administration). Furthermore, they had no history of diabetes or the use of any hypoglycemic agents. Thus, normal venous blood glucose levels documented at the time of low POCT glucose lead to a diagnosis of pseudohypoglycemia. The etiology in patient 1 was likely due to peripheral vascular disease. In patient 2, the cause was likely due to congestive heart failure, with poor peripheral perfusion.

Clinical Lesson: Low glucose levels are frequently encountered in clinical practice. It is essential to check if the non-diabetic patient is symptomatic or not. It is worth checking glucose levels from other sites in asymptomatic patients who do not fulfill Whipple's triad before preceding into an elaborate hypoglycemia work-up.

Diabetes Mellitus and Glucose Metabolism

DIABETES CASE REPORTS

Role of Octreotide in Sulfonylurea-Induced Hypoglycemia

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Introduction: The most common adverse effect associated with sulfonylurea ingestion is hypoglycemia. Sulfonylureas have very narrow therapeutic indices with a prolonged half-life in End-Stage renal Disease (ESRD). As per literature review, insulin and oral sulfonylureas are responsible for 13.9% and 10.7% of emergency hospitalizations respectively. It is, however, not surprising that intentional or unintentional overdose with these agents can lead to prolonged hypoglycemia which can prove to be fatal.

Case Report:76-year-old female presented to the Emergency Department (ED) with complaints of generalized weakness since the past three days. Her past medical history was significant for ESRD, Hypertension and Non-Insulin Dependent Diabetes Mellitus type II (home regimen of glipizide 10 mg daily). On physical exam, she was tachypneic and appeared lethargic. Her neurological exam was intact, and she was oriented to time, place and person. Her labs were significant for BUN of 77 mg/dL (5–20 mg/dL), Creatinine of 9.94 mg/dL (<1.3mg/dL) and blood glucose of 89 mg/dL (70-140mg/dL). Liver and thyroid

function tests were normal. Computed Tomography scan of the head was unremarkable. In the ED, she received 5 mg of glipizide after which she became more confused and lethargic. Her blood glucose level was 21mg/dL thus she received seven pushes of intravenous (IV) dextrose (25g each), two doses of intramuscular glucagon (1mg each) and was started on a continuous infusion of dextrose (D10) at 75cc/hour. Her blood glucose levels continued to remain low with a repeat value of 34 mg/dL and her mental status continued to worsen. Labs checked at that time were significant for a C-Peptide level of 22.13ng/ml (1.00–4.00ng/ml) and an insulin level of 43.7uU/ml (<20uU/ml) suggesting it to be sulfonylurea toxicity. Sulfonylurea level could not be checked due to laboratory limitations. She was started on subcutaneous octreotide 30 mcgs every 6 hours as per endocrinology recommendations. Her blood glucose started to improve, and her mental status returned to baseline. Per oral food intake was resumed, she remained euglycemic and octreotide was discontinued.

Conclusion: Octreotide is a synthetic octapeptide analogue of somatostatin which can effectively suppress insulin secretion. Glucose, on the other hand, would stimulate insulin release and cause rebound hypoglycemia. Boyle et al, showed that octreotide was superior to diazoxide and glucose in preventing sulfonylurea-induced hypoglycemia. Therefore, we as clinicians should be able to quickly recognize sulfonylurea toxicity as the cause of hypoglycemia and attempt to administer octreotide as soon as possible. This in turn would help decrease length of hospital stay and avoid the detrimental effects of hypoglycemia like seizures, coma and death especially in older individuals.

Diabetes Mellitus and Glucose Metabolism

DIABETES CASE REPORTS

SARS-CoV-2 Infection Related Diabetes Mellitus

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Introduction: SARS-Cov-2 (severe acute respiratory distress syndrome- coronavirus 2) viral infection has a predilection for pancreatic beta cells causing insulin deficiency. Studies from the SARS-CoV outbreak in 2003 highlighted the relationship between SARS-CoV and ACE-2 (angiotensin-converting enzyme 2) receptors in pancreatic islet cells. We describe a pediatric patient who developed Diabetes Mellitus after exposure to the Sars-CoV-2 virus.

Case Report: A previously healthy 13-year-old female of Mexican descent was found to be hyperglycemic at her annual visit. The patient endorsed polyuria and polydipsia for 3 weeks, and weight loss for 3months. 3 months prior to presentation, her mother became ill and tested positive for SARS-CoV-2 by PCR analysis. The patient had no SARS-CoV-2 associated symptoms.

Her exam was notable for a BMI was in the 78%ile for age with no acanthosis nigricans. She had no family history of diabetes or autoimmune disease.

Initial blood glucose was 729 mg/dL, with bicarbonate of 20.6 mEq/L, pH 7.45, and anion gap of 14 mEq/L. Large ketones were present in the urine. Her concomitant C-peptide level of 1.0 ng/ml was low in the setting of hyperglycemia. Her HbA1c was 14.3%. Diabetes-related autoantibodies, celiac, and thyroid antibodies were negative. Her Sars-CoV-2 antibody titer was positive with a negative PCR.

The patient was treated with a basal-bolus regimen of subcutaneous insulin at a maximal total daily dose of 0.7 u/kg/day. 5 weeks later, her insulin requirement and HbA1C were both lower; at 0.5 u/kg/day and 9.3% respectively.

Discussion: This patient's symptoms of hyperglycemia started shortly after her exposure to the SARS-CoV-2 virus. She had no features consistent with Type 2 DM. She similarly had no serological evidence of DM related autoimmunity, thus being different from reports of new-onset Type 1 DM with confirmed autoimmunity presenting during the Sars-CoV-2 pandemic. Although Type 1B DM without evidence of humoral islet autoimmunity and monogenic DM could not be fully excluded, we postulate that the patient developed SARS-CoV-2 associated DM given her time course and documented exposure to SARS –CoV-2 with the presence of SARS-CoV antibodies. One similar case has previously been reported By Holstein et al. [1] While we share the lack of direct evidence of causation, we postulate that more patients with similar presentations will be reported during the current pandemic.

Reference: 1.Hollstein, T et al. Autoantibody-negative insulin-dependent diabetes mellitus after SARS-CoV-2 infection: a case report [published online ahead of print, 2020 Sep 2]. *Nat Metab*. 2020;10.1038/s42255-020-00281-8. doi:10.1038/s42255-020-00281-8

Diabetes Mellitus and Glucose Metabolism

DIABETES CASE REPORTS

SARS-CoV-2 Trigger in Severe Insulin Resistance With Acute Haemolytic Crisis in Diabetes and Glucose-6-phosphate Dehydrogenase Deficiency

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Background: We report a case of severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) infection as trigger for increased insulin resistance and severe haemolytic crisis in a male with type 2 diabetes mellitus and glucose-6-phosphate dehydrogenase (G6PD) deficiency. **Clinical Case:** A 64-year-old man (BMI 25kg/m2, weight 75kg) with past medical history of type 2 diabetes mellitus (on metformin and sitagliptin; glycated haemoglobin 51 mmol/mol, n<42mmol/mol), hypertension, G6PD deficiency and gout was admitted to hospital with COVID pneumonitis and type 1 respiratory failure giving 5 days' history of