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Abstract #1184484

Transient Refractory Hyperinsulinemic Hypoglycemia

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**Introduction:** Fasting hypoglycemia in the setting of hyperinsulinemia typically is persistent and often progressive. We present a case where fasting hypoglycemia with hyperinsulinemia requiring multiple pharmacologic agents for correction was transient in nature over a fortnight. This occurred in the setting of recurrent seizure-like activity. Seizures are a well-known sequela of hypoglycemia; however, the reverse is not nearly as well documented.

**Case Description:** A 20 year old male with a history of developmental delay, chronic PEG tube, and nonverbal at baseline presented for breakthrough seizures after a 17 year seizure-free period. Although glucose was normal at 75 mg/dL on admission, in the ensuing days, his fasting glucose levels fell to less than 65 mg/dL. He required D10 infusion, and experienced return of hypoglycemia when D10 was stopped. Labs obtained during a hypoglycemic episode showed blood glucose 47 mg/dL, insulin 12 uU/mL, beta-hydroxybutyrate 0.07 mMol/L, C-peptide 7.9/mL, sulfonylurea screen negative, and negative insulin antibodies. Octreotide was started in addition to D10 to address persistent hypoglycemia. MRI abdomen did not reveal pancreatic lesions.

Over a two week period the D10 infusion was slowly weaned off. A 68 Gallium-DOTATAE PET scan was ordered and the octreotide was discontinued two days prior to the scan. The D10 infusion was stopped one day before the scan, and there was no return of hypoglycemia. The 68Ga-Dotate PET scan showed normal pancreatic structure and the patient was discharged home as his hypoglycemia resolved.

During his admission, the patient underwent EEG assessment, which did not show epileptiform activity but did show a moderate degree of diffuse or possibly multifocal cerebral dysfunction warranting clinical correlation

**Discussion**

True hypoglycemia needs to fulfill Whipple’s Triad: Documented hypoglycemia on venous sample, symptoms consistent with hypoglycemia, and resolution of symptoms with correction of hypoglycemia. In this case, the triad was presumed to be positive based on laboratory evaluation and a report of behavioral changes with hypoglycemia from patient’s parents. Appropriate laboratory assessment met criteria for endogenous hyperinsulinemia. Imaging which included 68Ga-DOTATAE PET did not reveal a source for excess insulin. He went from requiring D10 and octreotide to being euglycemic off both these medications.

Critical illness is also implicated in fasting hypoglycemia, particularly in end-organ failure or sepsis. The patient did not have any organ damage to this degree, but there was recurrent seizure-like activity. Status epileptics causes a massive release of catecholamines, which increases serum glucose. The latter then leads to a large insulin release from the pancreas, which can lead to a period of hypoglycemia. Although clear epileptiform activity was not noted on EEG, the patient’s clinical presentation with seizures raised the possibility that the hypoglycemia was related to the seizures. This case highlights the need to consider epileptiform activity as a cause of hyperinsulinemic hypoglycemia.

https://doi.org/10.1016/j.eprac.2022.03.116

Abstract #1184564

A 59-year-old man with type 2 diabetes mellitus with severe neuropsychiatric manifestations due to COVID-19 infection

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**Introduction:** Hyperglycemia in diabetes patients is associated with an increased risk for neuropsychiatric complications. SARS-CoV-2, COVID-19 is a serious pathogen with further exacerbation of neurological manifestations in patients with diabetes with a high risk for morbidity and mortality.

**Case Description:** We present a case of a 59-year-old man with poorly controlled type 2 diabetes (T2DM) for 16 years, dyslipidemia, and hypertension. His recent hemoglobin A1c is 7.9%. Past psychiatric history includes well-controlled anxiety and depression. He recently presented with a 19 infection and symptoms consistent with COVID-19. He has a history of past complications related to COVID-19 which required hospitalization. He has social, economic, and psychological burdens from his condition.

He required D10 infusion and experienced return of hypoglycemia when D10 was stopped. Labs obtained during a hypoglycemic episode showed blood glucose 47 mg/dL, insulin 12 uU/mL, beta-hydroxybutyrate 0.07 mMol/L, C-peptide 7.9/mL, sulfonylurea screen negative, and negative insulin antibodies. Octreotide was started in addition to D10 to address persistent hypoglycemia. MRI abdomen did not reveal pancreatic lesions.

Over a two week period the D10 infusion was slowly weaned off. A 68 Gallium-DOTATAE PET scan was ordered and the octreotide was discontinued two days prior to the scan. The D10 infusion was stopped one day before the scan, and there was no return of hypoglycemia. The 68Ga-Dotate PET scan showed normal pancreatic structure and the patient was discharged home as his hypoglycemia resolved.

During his admission, the patient underwent EEG assessment, which did not show epileptiform activity but did show a moderate degree of diffuse or possibly multifocal cerebral dysfunction warranting clinical correlation

**Discussion**

A literature review revealed variable presentation of neurological manifestations in patients with COVID-19 infection. T2DM patients have higher rates of anxiety and depression scores as well as a higher risk of developing Parkinson’s disease and Alzheimer’s dementia as compared to non-diabetic individuals. Studies suggest that in T2DM patients with long-term hyperglycemia, the coronavirus binds to angiotensin-converting enzyme -2 (ACE2) receptors which may exacerbate the development of...
neuropsychiatric disorders via direct tissue injury, viral entry to neuronal pathways, hypoxia, and immune response.

We conclude that patients with diabetes are susceptible to further brain injury with COVID-19 viral infection with a higher risk of morbidity and mortality. We stress the importance of COVID-19 vaccination in all diabetic patients.

https://doi.org/10.1016/j.eprac.2022.03.118

Abstract #1184571

Administering Nutritional Insulin Postprandially in the Inpatient Setting Is Associated with Improved Glycemic Metrics

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Objective: In many hospitals, as in the outpatient setting, nutritional (i.e. rapid-acting) insulin is typically injected before the meal. However, hospitalized patients frequently have poor oral intake due to their acute illness. Nurses must often guess whether to administer or withhold a patient’s preprandial insulin, depending on whether they feel the patient will eat. Guessing incorrectly can be deleterious, as administering insulin without food intake may result in hypoglycemia, while conversely holding insulin unnecessarily can lead to hyperglycemia. The objective of this study was to evaluate whether implementation of a hospital policy to deliver nutritional insulin post-prandially, as compared to pre-prandially, was associated with changes in inpatient glycemic metrics.

Methods: This was a retrospective cohort study performed at Howard County General Hospital, a member of Johns Hopkins Medicine, using a de-identified data set. In June 2019, hospital policy shifted the timing of all nutritional insulin to be given after at least 50% of the meal was consumed, instead of pre-prandially. Because a full-time endocrine hospitalist was hired to consult on all inpatients with diabetes in August 2018, we restricted the data set to compare the nine months pre-policy (September 2018 to May 2019) vs. nine months post-policy implementation (July 2019 to March 2020). Patients who received at least one unit of insulin and had a glucose measurement were included in the analysis. Primary outcome was the rate of inpatient hypoglycemia, as defined by a glucose ≤70 mg/dL. Secondary outcomes included rates of moderate hypoglycemia (≤54 mg/dL), severe hypoglycemia (≤40 mg/dL), hyperglycemia (mean daily glucose ≥180 mg/dL), length of stay (LOS), and 30-day readmission rates (30DRR). Chi-square with Yates’ correction or Student’s t-test were used to analyze the differences between groups.

Results: Rates of hypoglycemia significantly decreased from 5.9% (592 of 10,023 patient-days) to 5.0% (500 of 9,987 patient-days) post-intervention (p=0.006). Hyperglycemia rates also significantly decreased post-intervention (45.7% vs 42.5%; p<0.0001). Rates of moderate (1.9% vs 2.1%; p=0.23) and severe hypoglycemia (0.6% vs 0.6%; p=0.98) were not significantly different between groups. LOS and 30DRR were not significantly affected.

Discussion/Conclusion: Our study found that administering nutritional insulin post-prandially, instead of pre-meal, in the inpatient setting significantly decreased rates of hypoglycemia as well as hyperglycemia. Further study is warranted to evaluate whether this dosing strategy impacts patient experience or nursing comfort/distress regarding insulin dosing and inpatient diabetes.

https://doi.org/10.1016/j.eprac.2022.03.119

Abstract #1184598

Diabetic myonecrosis- An underreported and frequently misdiagnosed complication of diabetes mellitus

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Introduction: Diabetic muscle infarction (DMI), also referred as diabetic myonecrosis, is a rare and underdiagnosed complication of longstanding and poorly controlled diabetes mellitus. The usual presentation is sudden onset of pain at the involved muscles associated with swelling and tenderness. The diagnosis may be easily missed if clinical vigil is not high.

Case Description: A 40 year old male patient, known case of type 2 diabetes mellitus (T2DM) for 8 years presented with swelling and tenderness of the right thigh for past two weeks. Prior medical history was significant for hypertension and chronic kidney disease. The pain was sharp aching in nature and worsened with weight bearing or movement of affected limb. There was no prior history of trauma or fall. On examination, the right thigh was grossly swollen and tender to palpation. Complete blood count revealed presence of anemia (Hemoglobin 8.9 gm%) and neutrophilic leukocytosis(total leucocyte count 18,000/cu.mm). His current fasting plasma glucose(224mg/dl), post prandial plasma glucose(356mg/dl) and glycosylated hemoglobin(HbA1c) levels(11.9%, normal< 6.5%) were significantly elevated. Renal function tests were as follows: serum creatinine(2.4mg/dl, normal: 0.8–1.5), serum urea(71 mg/dl, normal:20–40), serum sodium(135 meq/l) and serum potassium(5.1 meq/l). Inflammatory markers like serum C-reactive protein (CRP) (28.9mg/dL, normal:1-5 )and erythrocyte sedimentation rate(80 mm/hr, normal:0-20 mm/hr) were significantly elevated. Serum creatinine phosphokinase(CPK) level(215IU/L, normal: 46-171) was raised. Blood culture revealed no growth. Chronic diabetic complications assessment revealed presence of moderate non-proliferative diabetic retinopathy, distal symmetrical polyneuropathy and chronic kidney disease. Doppler ultrasonography of bilateral lower limbs revealed presence of diffuse subcutaneous oedema of affected right thigh without evidence of deep vein thrombosis or significant occlusive atherosclerotic disease. Magnetic resonance imaging(MRI) of the right leg revealed presence of diffuse subcutaneous edema, intramuscular fascial edema and increased T2 signal intensity in affected muscle groups. A diagnosis of DMI affecting the right thigh was made. The patient was managed with insulin therapy for glycemic control, limb rest, judicious analgesic use and physical therapy resulting in improvement of his overall clinical status.

Discussion: Diabetic muscle infarction is a rare and serious complication seen in long-standing poorly controlled diabetes whose exact aetiopathogenesis remains poorly understood. It usually presents with the abrupt onset of pain, tenderness, and swelling of affected limb. The most common affected regions include thigh and calf muscles though other muscle groups may also be affected. The important differentials include muscle abscess, hematoma, deep vein thrombosis and myositis. MRI plays an