Diabetes Mellitus and Glucose Metabolism

DIABETES CASE REPORTS

The Troublesome Triad: Hypertriglyceridemia-Induced Acute Pancreatitis in Diabetic Ketoacidosis

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Background: Diabeticketoacidosis (DKA) is an acute metabolic derangement that can triggerhypertriglyceridemia-induced acute pancreatitis (HTGAP). While DKA classicallypresents in patients with Type 1 Diabetes Mellitus (DM1), previous literaturedescribes HTGAP in DKA as the initial presentation of Type 2 Diabetes Mellitus(DM2). The following case illustrates the diagnostic complexities of thisenigmatic triangle.
Clinical Case: Apreviously healthy, 28-year-old Hispanic male with a family history of DM2presented to the ED complaining of a sharp epigastric 10/10 pain radiating tothe right flank for one day that evolved into nausea with intractablenonbilious, nonbloody vomiting. He recently immigrated from Mexico City and hasnot seen a physician since childhood. He denied any tobacco, alcohol, norillicit substance use. Oninitial exam, he was afebrile and hemodynamically stable with dry mucosa,epigastric tenderness with voluntary guarding, but no evidence of jaundice,scleral icterus, nor abdominal distention. Pertinent initial labs foundglucosuria and ketonuria with a normal CBC, but his CMP was delayed as hisblood was lipaemic requiring serial dilutions that found hyperglycemia (415mg/dL, n <140), decreased carbon dioxide (17 mmol/L, n:23–29), a high aniongap (18 mmol/L, n<12), elevated ALT (154 u/L, n:4–36). Additional testingfound severely elevated triglycerides (4,427 mg/dL, n<150 mg/dL), elevated lipase(536 u/L, n<160), elevated Beta-hydroxybutyrate (5.30 mg/dL, n<2.81), andan elevated HgA1C (10.1%, n<5.7). These findings confirmed the diagnosis ofDKA with concerns for underlying HTGAP. CT abdomen showed hepatic steatosis andperipancreatic inflammation, confirming the diagnosis. Hewas admitted to the ICU, received aggressive IV fluid resuscitation with 5%dextrose in half normal saline while on an insulin infusion, with morphine andondansetron to control his pain and nausea. His abdominal pain resolved as histriglycerides trended down and anion gap closed. He was transitioned to an oraldiet with subcutaneous insulin prior to discharge on insulin and fenofibrate. At 1 month follow up, he reported medication compliance and denied anyrecurrence.
Conclusion: While DKA rarely triggers HTGAP in <4% of pancreatitis cases, HTGAP willexacerbate DKA as the pancreatic inflammation leads to acute beta celldysfunction causing insulin deficiency, propagating lipolysis. A serumtriglyceride level >1000 mg/dL is required for diagnosis of HTGAP, and thedegree of elevation is associated with the severity of pancreatitis.
Reference: Timilsina, S, et al. (2019). Triad of Diabetic Ket oacidosis,Hypertriglyceridemia, and Acute Pancreatitis: Severity of Acute PancreatitisMay Correlate with the Level of Hypertriglyceridemia. *Cureus, 11*(6), 4930–4934.

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Two Extremes of Dysglycemia- a Rare Case of Diabetes Complicated With a Non-Functional Pancreatic Neuroendocrine Tumor Transforming to an Insulinoma

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Background: Diabetes is characterized by hyperglycemia with heterogeneous pathophysiological features and varied presentation and consequences. Here, we present a rare case of diabetes that was complicated with insulinoma making glycemia management complicated.
Clinical Case: A 48-year-old white man was diagnosed with diabetes when he presented with weight loss, polyuria and polydipsia and HbA1c of 11%. He was treated with oral agents for few months without a good response then switched to insulin. He was assumed to have type 1 diabetes and had fairly well controlled glycemia on multi daily injection of insulin. He was diagnosed with a metastatic non-functional pancreatic neuroendocrine tumor (PTEN) 9 years later. He presented with epigastric pain and CT scan showed a mass in the pancreas tail and multiple lesions in liver. The tumor was immunopositive for S100, synaptophysin, and chromogranin. He was first treated with sunitinib and later switched to Everolimus. He underwent treatment with SIR-spheres which was complicated with post-embolization syndrome and Diabetes Ketoacidosis (DKA). He was switched back to Everolimus and referred to the endocrinology clinic for management of diabetes with a recent DKA. At the initial evaluation by the endocrinology team, he had BMI of 25 kg/m^2, no significant family history of diabetes, no diagnosis of dyslipidemia or hypertension. He had low C-peptide < 0.1 ng/mL (n: 0.8 - 5.2 ng/mL) and negative GAD antibody suggestive of non-immune mediated insulin deficient diabetes. He was treated with basal plus prandial insulin regimen and required about 0.6–0.7 unit/kg of insulin each day. Due to progression of PTEN, he was started on monthly Lanreotide while later Pembrolizumab was added resulting in hypothyroidism with a TSH of 75 mIU/L (n: 0.45 - 5.33 mIU/L) that was treated with Levothyroxine. Meanwhile, his diabetes care was complicated by recurrent hypoglycemic episodes and hypoglycemia unawareness. He gradually decreased the dose of insulin, stopped taking prandial insulin, and finally discontinued basal insulin due to recurrent hypoglycemia. He continued to have hypoglycemia despite stopping insulin. His C-peptide was found to be 3.3 ng/mL with a low BG of 62 mg/dl. Diazoxide was started and despite maximizing the dose, the patient continued experiencing hypoglycemia. He therefore decided to stop taking Diazoxide and only continued monthly Lanreotide. The patient is currently avoiding hypoglycemia by eating frequently (every 3 hours) and has regained his hypoglycemia awareness.
Conclusion: This is a rare case of non-immune mediated insulin deficient diabetes complicated by recurrent episodes of hypoglycemia due to a non-functioning PNET converting to a functional tumor producing insulin.