Diabetic ketoacidosis is an acute complication in diabetic patients precipitated by infection, inflammation, ischemia, or an iatrogenic cause.

Type B insulin resistant diabetes is an autoimmune disease, usually occurring with a concomitant rheumatologic disease.

Patients with Type B insulin resistant diabetes in DKA may require significantly increased amounts of insulin over several days before anion gap closure and resolution of ketonemia.

Case presentation

23 year old African American female with Type B insulin resistant diabetes and SLE presented to the ED with complaints of fatigue, decreased oral intake, and shortness of breath.

Found to have an anion gap metabolic acidosis, ketonuria, ketonemia, and hyperglycemia consistent with DKA.

Home insulin regimen: 1500 units of concentrated U500 regular insulin TIDAC + additional sliding scale.

Had received cycle 4/4 of treatment with rituximab and methylprednisolone two days prior to presentation.

Clinical course

Admitted to the ICU and started on an insulin drip and IV fluids.

HD 2: insulin drip rate over 200 units/hr

HD 4: developed steroid-induced acute and started on broad spectrum IV antibiotics for an axillary abscess. Insulin drip rate was over 500 units/hr, and although her anion gap had closed, ketonemia persisted.

HD8: maximum insulin drip rate of 1225 units/hr

HD 9: ketonemia resolved

HD 11: pharmacy procured U500 insulin for subcutaneous administration. Transferred to the general ward.

ANA 1:640
Anti-smith Ab >5.0
Anti-U1RNP Ab >240.0
Insulin antibody 7.0

Discussion

Type B insulin resistant diabetes is an exceedingly rare diagnosis occurring in the presence of a concomitant rheumatologic disease, most notably lupus, Sjogren’s, or mixed connective tissue disease.

This patient was felt to have developed insulin resistance in the setting SLE; Type B insulin resistant diabetes was diagnosed with elevated levels of IR autoantibodies and negative glutamic acid decarboxylase antibodies.

For Type B insulin resistant diabetics, the amount of insulin needed on a daily basis—and while in DKA—may be vastly increased due to the presence of insulin receptor (IR) autoantibodies, which must be out-competed by the insulin.

She underwent immunosuppression with four cycles of rituximab and methylprednisolone, which was thought to have triggered this episode of DKA. She was recently started on azathioprine in attempt to suppress her IR autoantibodies.

References


