

# Prolonged Diabetic Ketoacidosis with Hyperammonemia in the setting of Normal Liver Function

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**Background:** Acute encephalopathy in the setting of diabetic ketoacidosis is typically metabolic in origin, but less pursued are other causes including hepatic encephalopathy, namely hyperammonemia. These cases are less common in the setting of diabetic ketoacidosis and more so associated with cirrhotic pathology, however they should not be excluded in the differential diagnosis of patients with altered mentation in the setting of normal liver function.

**Case Report:** 37-year-old female patient presented to the emergency department with a chief complaint of confusion and nausea/vomiting over the past few days prior to admission. She has past medical history of type 1 diabetes mellitus, and she had intermittently taken her insulin over the past year. On initial presentation, patient was found to be in acute respiratory distress and ill-appearing. She was oriented to person but not place nor time.

On initial lab works, the patient had severe metabolic acidosis with pH less than 7 on venous blood gas and bicarb of 2 with anion gap of 30 and glucose elevated 581. Beta hydroxybutyrate was elevated on admission. Patient required emergent intubation given her severe respiratory distress. Surprisingly, her ammonium was elevated at 138 with normal liver function test.

Throughout her hospital course diabetic ketoacidosis protocol was followed and patient's anion gap closed within the three days. Patient was safely transitioned to subcutaneous long-acting insulin along with close follow-up with outpatient endocrinology. Liver function tests continue to remain stable throughout the hospital course.

**Conclusion:** This case highlights a rare manifestation of hyperammonemia in the setting of a young patient with normal liver and kidney function. It also highlights the molecular mechanisms behind diabetic ketoacidosis along with how they applied a clinical practice. Often overlooked are the protein catabolic reactions and their bioproducts that are underlying patients with prolonged ketosis, as seen in this case.