**Abstract for poster presentation**

Title: Unraveling the mystery of postoperative weakness: A case of euglycemic DKA after bariatric surgery.  
  
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Introduction:  
  
As the name suggest Euglycemic DKA is a condition with near normal glucose, PH< 7.3, serum bicarbonate <15 and ketosis. Euglycemic DKA is a relatively uncommon entity but it can be precipitated by starvation of any cause with concurrent stress. We report a case of euglycemic diabetic ketoacidosis precipitated by starvation in a patient with insulin dependent diabetes mellitus who underwent bariatric surgery and was possibly placed on a diet providing inadequate caloric intake.  
  
Case:  
  
A 63 year old Caucasian female with an extensive past medical history including super morbid obesity with a BMI of 50 presented with generalized weakness three days after a laparoscopic sleeve gastrectomy. She denied constitutional symptoms. The patient had no known nutritional deficiencies and had commenced a standard bariatric clear liquid diet on the second postoperative day. Her calorie intake was about 600-700 calories/day, protein intake of 60-70 gms, and fluid intake about 60 ounces/day as outlined by this diet.  
  
On presentation, her vital signs were within normal limits and the physical examination was grossly normal. Initial labs revealed a Hb of 15.5 mg/dl, BUN 41 mg/dl, creatinine 1.5mg/dl which was above her baseline compared to after her discharge a few days prior, glucose 200 mg/dl, lactic acid 1 mg/dl and urinalysis revealed more than 500 mg/dl glucose with ketones of 20 mg/dl. The blood gas was as follows: pH 7.26, pCO2 of 29, HCO3 of 13, PO2 of 91% on room air. She had an anion gap metabolic acidosis.  
  
The patient was initially managed with IVF and nutritional supplements believing that the cause of her symptoms was secondary to starvation ketoacidosis. However, she continued to decline clinically and repeat lab work one day later showed worsening anion gap metabolic acidosis as well as acetone qualitative level was reported as large and B hydroxybutyrate of 66.8 mmol/l. In light of this and now suspecting diabetic ketoacidosis, she was started on an insulin drip and transferred to the intensive care unit. Close monitoring of her BMP as per the DKA management protocol showed a gradual closure of her anion gap and the patient’s symptoms improved. She was discharged after 2 days on a diet with a higher caloric intake and had no untoward event.  
  
Discussion:  
  
It can be challenging to differentiate DKA from starvation ketoacidosis in patients who are diabetic and have been exposed to limited calorie intake. These patient usually have normoglycemia but urine shows ketones. The explanation is that glycogen depletion, lipolysis and free fatty acid production can result in normoglycemia with metabolic acidosis during the fasting phase. During starvation free fatty metabolism will convert the glucose to glycogen through gluconeogenesis. This leads to decrease insulin which stimulates gluconeogenesis causing normal or even low blood glucose levels which is contrary to the traditional findings of hyperglycemia in DKA. Unlike our patient who on presentation had near normal sugars but the level of bicarbonate below 18 were more indicative of DKA over starvation .  
  
There can be a significant overlap in the clinical and biochemical presentation of the metabolic acidosis caused by starvation as well as that of a DKA in the face of euglycemia. This can create an arduous task in making the diagnosis and more so, initiating the correct management. It is important to understand the pathophysiology of both entities particularly when presented with a patient with diabetes in order to tailor the management and achieve clinical improvement.