A Case of Lactation Ketoacidosis Followed by Striking Hyperglycemia

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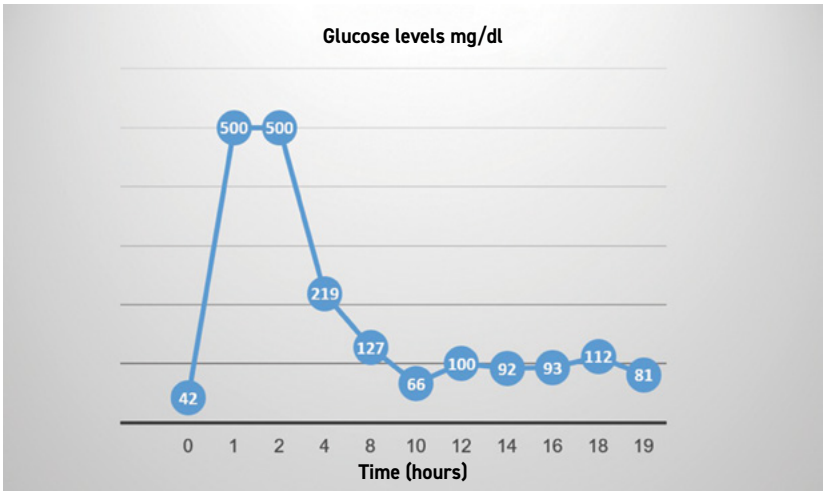
Lactation ketoacidosis (LA) is a well-documented phenomenon in cows, known as bovine ketoacidosis. It occurs when there is a negative energy balance because of a high glucose demand for milk production. LA in humans is rare, although the first case was reported in 1982 [1] and several cases have been reported in recent years. In most cases, there is a recognizable precipitating factor, often fasting or consuming a low calorie or low carbohydrate diet. However, a case of recurrent LA with no apparent trigger has also been reported [1]. In most cases the patients were euglycemic on presentation; however, hypoglycemia had been reported [2]. In this study, we present a case of LA and hypoglycemia on presentation with an unexpected striking hyperglycemia after treatment with dextrose.

PATIENT DESCRIPTION

A 33-year-old female presented to the emergency department (ED) with 4 days of nausea and vomiting, during which she consumed extremely little food. She had one episode of diarrhea and received 1000 cc of intravenous fluids at home. She was 4 months after her sixth delivery and was lactating. She had no history of chronic illnesses, and her pregnancies were uneventful. She was a nonsmoker, did not consume alcohol and was not taking medications. She consumed a low-calorie plant-based diet regularly.

On her arrival, she was severely fatigued. Her pulse was 116 bpm, her blood pressure was 113/75 mmHg, and her temperature was 36.7°C. Her blood tests demonstrated hypoglycemic ketoacidosis with glucose level of 44 mg/dl, pH 7.21, pCO_2 28 mmHg, HCO_3 11.2 nmol/l, and beta ketones 3.5 nmol/l. She received 2000 cc of D5%+NaCl 0.9% fluids, and her serum glucose levels increased rapidly to over 500 mg/dl. The dextrose was switched to 3000 cc of NaCl 0.9%. Approximately 20 hours later, she felt better and resumed normal eating. The acidosis resolved, and her serum glucose was normal. Five days later, she was well, her glucose level was normal, her HbA1c was 4.7%, and anti-glutamic acid decarboxylase (GAD) antibodies were negative [Figure 1].

Figure 1. Chronology of glucose levels from presentation through recovery



COMMENT

In recent years, several cases of LA have been reported [1]. Lactation is

energy demanding and when accompanied with a low calorie and low carbohydrate diet may cause a shortage in metabolic requirements and increase the usage of ketones for energy [3]. Over time, ketosis becomes more prominent and may transform into ketoacidosis [4]. Most cases are euglycemic due to enhanced glycogenolysis and gluconeogenesis. However, markedly reduced carbohydrate intake may cause hypoglycemia [3].

In our case, the patient had consumed extremely little food for 4 days, hence the hypoglycemia. The treatment for LA is based on intravenous dextrose, stimulating the secretion of insulin and suppressing glucagon. This combination reduces ketogenesis and acid formation [4]. In our case, the patient developed an unexpected striking hyperglycemia that exceeded 500 mg/dl and persisted for over 4 hours despite cessation of dextrose. Hyperglycemia during dextrose infusion for LA has been reported [3]. However, we are unaware of such prominent hyperglycemia. The onset of type 1 diabetes was excluded by the spontaneous resolution of the hyperglycemia and negative anti-GAD antibodies.

The refeeding syndrome comprises various metabolic changes that occur during malnutrition treatment, including hypophosphatemia, hypokalemia, hypomagnesemia, and hyperglycemia [5]. This condition is attributed to the increase in insulin following carbohydrate consumption. The hyperglycemia is explained by insulin resistance that prevails simultaneously. We suggest that the hyperglycemia in our case may be attributed to a similar insulin resistance. Alternatively, it may involve a shutdown of insulin secretion, and a transient mitigated glucose induced hyperinsulinemia because of the low intake and the vomiting as occurs in starvation ketoacidosis [1]. Unfortunately, the levels of insulin were not tested precluding a definite conclusion.

CONCLUSIONS

We present a case of LA with hypoglycemia on presentation followed by severe hyperglycemia in response to dextrose treatment. The hypoglycemia may represent severity but also constitutes a risk factor for hyperglycemia and therefore warrants careful treatment with dextrose in similar cases.

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Capsule

Physical activity as a modifiable risk factor in preclinical Alzheimer's disease

Physical inactivity is a recognized modifiable risk factor for Alzheimer's disease (AD), yet its relationship with progression of AD pathology in humans remains unclear, limiting the effective translation into prevention trials. Using pedometer-measured step counts in cognitively unimpaired older adults, **Yau** et al. demonstrated an association between higher physical activity and slower cognitive and functional decline in individuals with elevated baseline amyloid. Importantly, this beneficial association was not related to lower amyloid burden at baseline or longitudinally. Instead, higher physical activity was associated with slower amyloid-related inferior temporal tau accumulation, which significantly mediated the association with slower cognitive decline. Dose-response analyses further revealed a curvilinear relationship, where the associations with slower tau accumulation and cognitive decline reached a plateau at a moderate level of physical activity (5001–7500 steps per day), potentially offering a more approachable goal for older sedentary individuals. Collectively, these findings support targeting physical inactivity as an intervention to modify the trajectory of preclinical AD in future prevention trials, and further suggest that preferentially enrolling sedentary individuals with elevated amyloid may maximize the likelihood of demonstrating a protective effect of physical activity on tau accumulation and cognitive and functional decline in early AD.

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