SAGE Open Medical Case Reports
Volume 5: 1–2

DOI: 10.1177/2050313X17700743
journals.sagepub.com/home/sco
SAGE

Euglycaemic ketoacidosis in a non-diabetic primigravida following an appendicectomy

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Abstract

Pregnancy creates significant alterations in energy metabolism which itself is a physiological adaptation to provide continuous flow of energy metabolites to the foetus. The state of insulin resistance created by hormonal changes in pregnancy enables free flow of glucose to the foetus and allows its absorption through facilitated diffusion. As glucose is preferentially available for the foetus, maternal fasting glucose level would be less than that of a non-pregnant state and in contrast plasma ketones and free fatty acids levels are elevated, resulting in a state of accelerated starvation. These metabolic alterations place a pregnant woman at a higher risk of developing euglycaemic ketoacidosis when allowed to fast for prolonged periods due to medical, surgical and psychological reasons. We report a rare case of euglycaemic ketoacidosis causing severe increased anion gap metabolic acidosis in a non-diabetic mother following surgery for appendicitis at a gestation of 27 weeks.

Keywords

Euglycaemic ketoacidosis, pregnancy

Date received: 16 July 2016; accepted: 23 February 2017

Introduction

Euglycaemic ketoacidosis is a condition characterized by accelerated ketogenesis in cellular level in spite of adequate supply of glucose for energy metabolism, in contrast to diabetic ketoacidosis where there is intracellular glucose depletion resulting in accelerated ketogenesis providing keto acids as an alternative energy metabolite. The hormonal changes that occur in pregnancy create a state of insulin resistance allowing free flow of glucose to the foetus. Thus, prolonged starvation in a pregnant woman will place her at high risk of starvation ketosis. We describe a 27-year-old non-diabetic primigravida woman who presented with increased anion gap metabolic acidosis secondary to starvation ketoacidosis following prolonged fasting and vomiting due to appendicitis.

Case history

A 27-year-old primigravida woman presented at her 27th week of gestation with generalized abdominal pain, loss of appetite and vomiting for 1 day duration to a district general hospital. Initial obstetric evaluation was focused on excluding possibility of an obstetric emergency. An ultrasound scan of abdomen performed was unremarkable. On day 3 of the illness, her abdominal pain worsened; she was more tachypneic and rebound tenderness was elicited over right iliac fossa. A clinical diagnosis of acute appendicitis was made and she underwent a diagnostic laparoscopy on the same day itself. Laparoscopic findings were consistent with gangrenous appendix with caecal involvement. Subsequently, she underwent an open appendicectomy. Following surgery, she was found to be unwell and persistently tachypneic; however, her haemodynamic status was stable and the arterial blood gas (ABG) analysis showed compensated metabolic acidosis. Patient was transferred to Teaching Hospital Jaffna, the tertiary care centre of the region, for further evaluation and management. On arrival to intensive care unit (ICU), her capillary blood sugar was 99 mg/dL and ABG

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done on ambient air revealed severe metabolic acidosis with pH 7.2, pCO_2 18 mmHg, HCO_3^- 7 mmol/L and BE −21.0 mmol/L. Serum electrolytes on admission to ICU were Na^+ – 136 mmol/L, K^+ – 3.3 mmol/L and Cl^- – 100 mmol/L. She had persistently low lactate level with normal liver biochemistry and renal profile which made sepsis a remote possibility to cause severe increased anion gap metabolic acidosis. She was treated with intravenous $NaHCO_3$ to prevent detrimental effect of severe metabolic acidosis on foetus; however, further metabolic workup related to acidosis showed a positive Rothera's test for serum as well as for urine which were supportive of starvation ketoacidosis. She was started on intravenous Ringer's lactate with 50% dextrose and intravenous potassium replacement. Patient made a remarkable recovery with intravenous dextrose and was started on enteral feeding after discussing with treating surgical team.

Discussion

Pregnancy is characterized by complexed metabolic and endocrine adaptation processes including impaired insulin sensitivity, increased β cell response, moderately increased blood glucose levels and changes in circulating free fatty acid levels, triglycerides, cholesterol and phospholipids in order to meet the energy demands of the foetus. These metabolic adjustments predispose to severe ketoacidosis even in the absence of diabetes mellitus during pregnancy. Furthermore, the ketotic tendency is augmented by the state of respiratory alkalosis created by central respiratory stimulation by progesterone resulting in lower plasma bicarbonate concentration causing reduced buffering capacity.[1]

Metabolic acidosis of any aetiology is alarming in pregnancy as it can lead to adverse foetal and maternal outcomes. Adverse effects on foetal neurodevelopment, level of intelligence and even foetal demise are inevitable if metabolic acidosis is not promptly addressed during pregnancy.[2] Furthermore, acute severe metabolic acidosis in pregnancy will also have negative impact on maternal cardiovascular stability; it can lead to maternal hypotension due to reduced cardiac contractility, vasodilatation and also due to ventricular arrhythmias induced by acidosis. Pregnancy itself is an immunosuppressed state; hence, the uncorrected metabolic acidosis would cause more detrimental effects on leukocyte function leading to higher risk of maternal infections and indirectly foetal as well.[3]

In our case, the patient had severe acidosis (pH 7.2) on arrival to ICU with bicarbonate level of 7 mmol/L; however, serum lactate level was normal which made the diagnosis of lactic acidosis unlikely. Diabetic ketoacidosis was unlikely without prior history of diabetes and normoglycaemia on admission. Patient denied any history of eating disorders like anorexia and any history of toxin ingestion. She was normotensive and had normal transaminase levels with normal platelet count making both preeclampsia and Haemolysis Elevated Liver Enzymes and Low Platelets (HELLP) syndrome unlikely.

Initial vomiting at presentation and extensive fasting perioperatively as well as postoperatively would have precipitated accelerated starvation in our patient resulting in severe ketoacidosis causing increased anion gap metabolic acidosis.

Euglycaemic ketoacidosis precipitated by accelerated starvation in pregnancy should be considered as an important differential diagnosis for acidosis in pregnancy. Avoiding or minimizing the periods of fasting in pregnant mothers, prompt recognition of starvation ketoacidosis and early correction with intravenous dextrose will prevent foetal as well as maternal morbidity and mortality.

Declaration of conflicting interests

The author(s) declared no potential conflicts of interest with respect to the research, authorship and/or publication of this article.

Ethical approval

Ethical approval to report this case report was obtained from the Jaffna Medical Faculty Ethical Review Committee, Sri Lanka (ERC/35/MF/2016).

Funding

The author(s) received no financial support for the research, authorship and/or publication of this article.

Informed consent

Informed written consent was obtained from the patient for her anonymized information to be published in this article retrospectively.

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