Case Report

Diabetic Ketoacidosis during Pregnancy

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ABSTRACT

Diabetic Ketoacidosis is life is a life threatening complication of diabetes, characterized by hyperglycemia, ketoacidosis and ketonemia. We present a case of 28 year old female with 4-month pregnancy admitted in ICU with vomiting for 2 weeks, fever and breathlessness for 2 days and irritability and abdominal pain for 1 day. Random blood sugar was 428 mg/dl and urine ketones 3+. There was no previous history of Diabetes. She was diagnosed as a case of diabetic ketoacidosis.

KEY WORDS. Pregnancy, Diabetes Mellitus, Ketoacidosis.

INTRODUCTION

Daibetic Ketoacidosis (DKA), is life threatening complication of diabetes, especially if it occurs during pregnancy. The reported incidence of DKA outside of pregnancy ranges from 4.6 to 8 episodes per 1000 patients annually1. Numerous review articles and retrospective studies have found the incidence to range from 1% to 10% during pregnancy.

Cousins2 reported the incidence of DKA during pregnancy to be 9.3% in a group of 1508 patients studied between 1965 and 1985. More recent retrospective studies by Rodgers and Rodgers3 and Cullen and colleagues4found an incidence of DKA in pregnancy of 1% to 2%. A case series by Kilvert and colleagues[5], the reported incidence of DKA among 635 pregnant patients who had pregestational type 1 diabetes mellitus was 1.73%. A larger, more recent case series by Schneider and colleagues[6] reported the incidence of DKA in pregnant patients to be 1.2% among women who received insulin for diabetes control during their gestation.

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In one study, 30% of cases of DKA occurred in women who did not have known diabetes[7].

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A study by Drury and colleagues[9] found the maternal mortality rate to be less than 1% among 13 episodes of DKA experienced in 600 consecutive pregnancies. Fetal loss rates, however, are much higher. The study by Drury and colleagues[9] reported a fetal mortality rate of 85%.

Montoro and colleagues7 reported a 35% incidence of fetal demise in women who had type 1 diabetes mellitus who presented with DKA.

The fetal mortality rate was even higher (57%) in the one third of patients for whom the episode of DKA was their first diagnosis of diabetes.

CASE REPORT

A 28 year old female was admitted in ICU with history of 4 month pregnancy, 2 week history of vomiting, 2 day history of fever & breathlessness and 1 day history of abdominal pain & restlessness. On examination she was dyspnec, tachycardiac and dehydrated. Her chest was clear.

GCS was 12/15 and there was no focal neurologic deficit.

Abdomen was tender all over and there was no visceromegaly apart from gravid uterus.

There was no previous history of Diabetes and Hypertension.

All base line investigations were sent. Random blood sugar was 428mg/dl and urinary ketones were 3+. Physician on call diagnosed her as a case of diabetic ketoacidosis and she was instituted on insulin infusion and fluid therapy. To relieve abdominal pain Inj: Nalbuphine 10 mg diluted was given I/V slowly. After a few minutes patient's consciousness worsened. ICU team was called and patient was shifted to ICU for further management and monitoring.

During treatment she developed vaginal bleeding and she was shifted to Operation Theater and pregnancy was terminated through LSCS.She recovered uneventfully.This case is important as there was no past history of Diabetes and the use of opiod analgesic compromised the respiratory mechanism to compensate for metabolic acidosis. This also emphasizes the importance that young pregnant women with such presentation should be considered as a probable case of DKA, when other possibilities are ruled out.

DISCUSSION

Diabetic ketoacidosis is a serious complication of Diabetes. It increases the risk maternal & fetal mortality and morbidity when it occurs during pregnancy.

Pregnancy is a relatively diabetogenic state. It is well known that overall insulin resistance and lipolysis are increased during normal pregnancy; the increased lipolysis contributes to the "accelerated starvation" and propensity toward ketone body formation during pregnancy10. Hormones such as human placental lactogen (HPL),growth hormone, prolactin, and progesterone play key roles in insulin sensitivity key adaptation of pregnancy that contributes to a propensity to DKA involves the intricate link between the renal and respiratory systems. Increases in minute ventilation at the alveolar level place the pregnant woman in a state of respiratory alkalosis. At the renal level, this is compensated by increased excretion of bicarbonate, a key metabolic buffer. This state of "compensated respiratory alkalosis" during pregnancy plays its role by decreasing the pregnant woman's ability to buffer ketone acids present in the serum during episodes of DKA.

The clinical hallmarks of DKA (hyperglycemia, hypovolemia, ketosis, and acidosis) are the result of an exaggerated counter-regulatory response to the perceived hypoglycemia, which sets off a cascade effect that becomes apparent in the clinical presentation and laboratory findings. Insulin counter regulatory hormones such as glucagon are released into the circulation in response to cellular hypoglycemia, causing gluconeogenesis and glycogenolysis to become disinhibited at the level of the liver. Therefore, the hyperglycemia in DKA originates from three sources: (1) a high availability of glucose precursors due to glucagon and epinephrine driven lipolysis (glycerol) and muscle breakdown (amino acids); (2) a breakdown of glycogen stores; and (3) a decreased peripheral uptake of glucose, caused by insulin lack and made worse by increased counter-regulatory hormones. The increased insensitivity to insulin results in decreased adipocyte storage of free fatty acids, now present in the circulation in high amounts due to increased lipolysis. These increased fatty acids undergo oxidation and are converted to ketoacids by the liver (3-b-hydroxybutyrate and acetoacetate). The ketoacid acetoacetate may undergo decarboxylation and conversion to acetone, and can often present clinically as a fruity odor from the patient's breath11 . The increased levels of ketone bodies, combined with the buildup of lactic acid from peripheral hypoperfusion, result in the metabolic acidosis seen with DKA.

Many studies have been performed on precipitating causes of DKA in hopes to better recognize and prevent the onset of this disease process. Rodgers and Rodgers[3] looked at variables associated with DKA in pregnancy by retrospectively reviewing admissions of affected patients over a 10-year period. These data were then compared with the existing literature regarding DKA in pregnancy, for a total of 64 cases. The most common precipitating event was emesis from any cause, accounting for 42% of DKA cases in their study. The second most common precipitating event was use of beta sympathomimetics, and when

with emesis, these events accounted for 57% of episodes of DKA in this study. Other contributing variables included infection, poor patient compliance, insulin pump failure, undiagnosed diabetes, and physician management errors. A total of 80% of DKA episodes in this study could be attributed to beta sympathomimetics, emesis, poor compliance, and physician management errors. In a similar study, Montoro and colleagues[7] found that poor patient compliance was the most common variable inciting episodes of DKA. Cessation of insulin use in the study population accounted for 35% of DKA episodes, whereas infection accounted for 20%. Based on these studies, seven general factors can be associated with precipitating the onset of DKA during pregnancy: emesis, infection, poor compliance/noncompliance, insulin pump failure, use of beta sympathomimetics, use of corticosteroids, and poor physician management.

The laboratory findings seen inDKA can be used to help confirm correct diagnosis of the disease. Finding of hyperglycemia, acidosis, and ketonemia are generally seen in all cases of DKA12. Plasma glucose levels are usually well over 300 mg/dL, but episodes of DKA in pregnancy can be seen at much lower glucose level s in pregnancy.

Blood glucose levels less than 200 mg/dL have even been reported in some cases of DKA during pregnancy[4].

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