Acta Scientific Women's Health (ISSN: 2582-3205)

Volume 2 Issue 2 February 2020

Case Report

Diabetic Ketoacidosis in Pregnancy: A Rare but Serious Threat to Mother and Fetus

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DOI: 10.31080/ASWH.2020.02.0083

Received: December 20, 2019

Published: January 22, 2020

Abstract

Introduction: Diabetic ketoacidosis (DKA) is a serious metabolic complication of diabetes with high mortality if undetected. Its occurrence in pregnancy compromises both the fetus and the mother profoundly. Fortunately, the occurrence of DKA in women with diabetes who become pregnant is rare ~ 1-3%. Pregnant women are at a greater risk for DKA than are non pregnant diabetic women.

Clinical Description: We present a case of a 23 year old patient G2P1L1 with 37.2 week period of gestation, a known c/o Type 1 diabetes mellitus, referred from another hospital with sinus tachycardia. On proper examination and investigation, patient was diagnosed a case of diabetic ketoacidosis in pregnancy. Patient was admitted in high dependency unit (HDU) and was managed according to the protocol. Patient was put on continuous cardiotocography (CTG). CTG was showing fetal tachycardia with absent variability. Patient was taken up for category 1 caesarean section. Baby delivered was deeply cyanosed with no fetal heart beat. Resuscitative efforts were accomplished in the form of cardiopulmonary resuscitation, vasopressors, oxygen. Despite best efforts baby could not be revived. Arterial blood gas analysis of cord blood revealed acidosis. Post caesarean patient was shifted to HDU and discharged on day 4 in satisfactory conditions.

Discussion: A single episode of DKA poses considerable risk to the fetus. In our case CTG showed absent variability and the outcome was a fresh still birth with ABG showing acidotic fetus. Kamalakannan D (2002) also reported an adverse fetal event ~ a still birth at 36 weeks POG to a 28 year old female with type 1 Diabetes complicated with ketoacidosis. Another episode of fetal demise had been reported by Carrol MA (2005) at 31 weeks of POG to a 23 year old woman G3P2. 5% fetal mortality rate have been reported by Baagar KA (2017) in their 3 year retrospective study on analysis of diabetic ketoacidosis in pregnant women.

Conclusion: Prevention, early recognition, hospitalisation and aggressive management remain the cornerstones to minimise the outcomes of this dreaded complication.

Keywords: Cardiotocography (CTG); High Dependency Unit (HDU); Diabetic Ketoacidosis (DKA)

Introduction

Diabetic ketoacidosis is a serious metabolic complication of diabetes with high mortality if undetected. Its occurrence in pregnancy compromises both the fetus and the mother profoundly. Although predictably more common in patients with type 1 Diabetes, it has been recognised in those with type 2 diabetes as well as gestational diabetes, especially with the use of corticosteroids for fetal lung maturity and beta 2 agonists for tocolysis [1-3]. Diabetic ketoacidosis usually occurs in the second and third trimesters because of increased insulin resistance, and is also seen in newly presenting type1 Diabetes patients [4].

Fortunately, the occurrence of diabetic ketoacidosis in women with diabetes who become pregnant is rare ~1-3% [5,6]. Diabetic

ketoacidosis is rarely life threatening to the pregnant women if it is recognised and treated promptly. The risk of maternal mortality secondary to complications from DKA is not well established [7]. However, fetal loss rates remain in the order of 10-25% for a single episode of diabetic ketoacidosis despite substantial improvements in perinatal and neonatal care [8,9]. According to Sibai BM, DKA in pregnancy (DKP) associated fetal mortality ranges from 9% to 35%, while maternal mortality is < 1% [10].

Compared with diabetic ketoacidosis in non pregnant patients, DKP is unique in a number of different aspects; it usually happens at lower (or even normal) blood glucose levels 11 and progresses rapidly, requiring prompt diagnosis and management.

Case Report

Patient 23 year old G2P1L1 with 37.2 weeks period of gestation, a known case of Type 1 Diabetes Mellitus since 6 years of age, referred from another hospital with complaint of sinus tachycardia. On arrival her vitals were BP: 134/84 mm of Hg, PR: 134/min, O_2 saturation 98%, RR: 24/min. On CTG fetal heart rate was showing fetal tachycardia. On examination she was tachypnoeic, hyperventilating and dehydrated. Initial laboratory work up confirmed hyperglycaemia (RBS- 310 mg/dl), blood urea -4.8 mmol/L, creatinine-114.74 mmol/L and high total leucocyte count of 21.53×10^3/cmm, serum sodium of 137 mmol/L, potassium-3.7 mmol/L, chloride -105 mmol/L. Immediately patients Arterial Blood Gas analysis was done. It revealed acidosis. pH =6.84, pCO_2 =21.4 mm Hg, Base deficit of 27.2mmol/l, HCO_3 =5.1 mmol/l. Urinalysis revealed sugar 2 ++, ketones 2++. Patient was diagnosed to be a case of Diabetic Ketoacidosis.

She was admitted to high dependency unit, resuscitated with supplemental oxygen, I/V fluids and insulin infusion as per the protocol for diabetic ketoacidosis. She was put on left lateral position to release aorto caval compression. Potassium chloride was added to intravenous fluids to correct serum potassium levels. Patient was given one ampoule of Bicarbonate (50 meq).

Patient was put on continuous CTG. CTG was showing decreased variability and fetal tachycardia. After maternal stabilisation and reversal of acidosis patient was assessed for Bishop scoring. Score was 2. Patient was taken up for emergency Caesarean section in view of suspicious CTG with no variability. Baby delivered was cyanosed with no fetal heart beat. Baby was intubated and given oxygen by ambu bag, but no improvement, Cardiopulmonary resuscitation done in the form of cardiac compressions and inotropic drugs such as adrenaline. There was no improvement. CPR continued for next 30 min and finally declared dead. It was a fresh still birth with bwt 3.85 kgs. On Arterial Blood Gas analysis of the cord (venous) blood: pH=6.71, pCO_2= 36.3 mmHg, Base deficit = 27.4 mmol/L.

Post caesarean patient was shifted to the high dependency unit and managed according to the protocol. Patient general condition improved and patient was shifted from HDU to ward on day 2. Finally patient got discharged from the hospital on day 4 and asked to follow up after a week as outpatient basis.

Discussion

Pregnant women are at greater risk for DKA than are non pregnant women [11]. Factors that predispose the pregnant women to DKA include accelerated starvation, (especially in the second and third trimester), dehydration, decreased caloric intake (e.g. nausea or hyperemesis gravidarum), decreased buffering capacity (compensated respiratory alkalosis of pregnancy), stress and increased production of insulin antagonists (human placental lactogen, prolactin and cortisol) [12]. The most common precipitating risk factors for the development of DKA (as in non pregnant patients) are infection related acute illness (viral/bacterial ~30%) and failure to take insulin as prescribed (usually due to patient non compliance ~ 30%) [13] Rodgers and Rodgers found that emesis and use of beta –adrenergic medications were considered etiologic in 57% of cases of DKA, while patient non compliance and physician management errors were etiologic errors in 24% and contributory in 16% of DKA cases in their retrospective review [14].

DKA poses an immediate threat to maternal well being. Severe dehydration can lead to hypotension, acidosis can cause organ dysfunction and electrolyte imbalance can cause cardiac arrhythmias [15]. Maternal hyperglycemia results in fetal hyperglycemia and fetal osmotic diuresis. The fetus can also become acidotic from keto acids that cross placenta. Acidemia decreases uterine blood flow, reduces tissue perfusion, and leads to decreased oxygenation of the feto placental unit. Further more, a leftward shift of the maternal oxy haemoglobin dissociation curve with decreased 2,3-diphosphoglycerate increases haemoglobin affinity for oxygen decreasing fetal oxygen delivery [6,12,14].

Because the fetus is not directly accessible, inferences regarding fetal status are often made from the external recording of the fetal heart rate. Often decreased or absent variability, absent accelerations, and late decelerations as observed on external fetal heart rate tracings with decompensated maternal DKA [15]. Doppler Ultrasound has also been used to look at blood flow in fetal vessels with DKA. Transient fetal blod flow redistribution was demonstrated in the umbilical and Middle Cerebral Artery as measured by Pulsatility index. Reversal of the abnormal flow was demonstrated after treatment of maternal DKA [16].

A single episode of DKA poses considerable risk to the fetus. In our case CTG showed absent variability, and the outcome was a fresh still birth, a deeply cyanosed with ABG showing acidotic fetus. Kamalakannan D (2002) also reported an adverse fetal event ~ a still birth at 36 weeks POG to a 28 year old female with type 1 Diabetes complicated with ketoacidosis [4]. Another episode of fetal demise had been reported by Carrol MA (2005) [15] at 31 weeks of POG to a 23 year old woman G3 P2. 5% fetal mortality rate have been reported by Baagar KA (2017) [17] in their 3 year retrospective study on analysis of diabetic ketoacidosis in pregnant women.

Conclusion

While the outcomes of Diabetic ketoacidosis in pregnancy have improved a lot since the past few years, significant fetal mortality still remains. Prevention, early recognition, hospitalisation and aggressive management remains the cornerstone to minimise the outcomes of this dreaded complication.

Acknowledgement

The authors reported no conflict of interest and no funding was received for this work.

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