Diabetic Ketoacidosis In Newly Diagnosed Gestational Diabetes With Type 2 Diabetes Postpartum

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Abstract

Diabetic ketoacidosis is a rare complication of gestational diabetes. We report a case of a previously healthy woman with an uncomplicated pregnancy, presenting in the third trimester with diabetic ketoacidosis and who was diagnosed with type 2 diabetes postpartum.

INTRODUCTION

Diabetic ketoacidosis is a rare complication of gestational diabetes with significant maternal and neonatal morbidity and mortality. Although more common in patients with type 1 diabetes, it has been described in those with type 2 diabetes as well as gestational diabetes, especially with the use of corticosteroid and -adrenergic agonists for premature labour.[1,2,3] Therapy is directed toward aggressive correction of fluid imbalance, replacement of electrolytes, restoration of glucose homeostasis and treatment of precipitating factors in an intensive care setting. We describe a woman with an uncomplicated pregnancy presenting in the third trimester with diabetic ketoacidosis and who was diagnosed with type 2 diabetes postpartum.

CASE REPORT

A previously healthy primigravid 38 years old Black African woman presented at 30 weeks and five days of gestation for the first time at our unit, having booked for antenatal care elsewhere, complaining of three days history of vomiting, back pain and reduced fetal movements. On direct questioning, she also has polyuria and polydypsia. There was no family history of diabetes. On examination, she was tachypnoeic, hyperventilating, tachycardic and dehydrated but afebrile. Abdominal examination was otherwise normal for the gestational age.

Venous plasma glucose was 28 (<7.8) mmol/L, sodium 128 (135-145) mmol/L, potassium 5.6 (3.5-5.0) mmol/L, urea 6.6 (3.3-6.7) mmol/L, creatinine 135 (68-106) ?mol/L. Arterial blood gas showed severe metabolic acidosis pH 7.16 (7.35-7.45), carbon dioxide tension 1.3 (4.7-6.0) kPa, oxygen tension 16.6 (>10.6) kPa, bicarbonate 3.4 (22-28) mmol/L, base excess -25 (2) mmol/L and lactate 2.7 (0.6-1.7) mmol/L. Urinalysis showed 3+ ketones, 4+ glucose, 2+ protein and 3+ blood. Glycated haemoglobin (HbA1c) was 10% (<6.5%) and fructosamine 396 (<240) ?mol/L.

She was admitted to intensive care unit and resuscitated with intravenous fluids and insulin sliding scale infusion as per regime for diabetic ketoacidosis. Intravenous antibiotic was also commenced for possible urinary tract infection, which was later confirmed to be a coliform infection. Once the metabolic acidosis was corrected, she was treated with basal bolus subcutaneous insulin regime.

Ultrasound scan for fetal growth at 31 weeks and one day showed polyhydramnios (amniotic fluid index [AFI] of 23cm) with a macrosomic fetus (abdominal circumference well above 90th centile and head circumference on the 50th centile) consistent with poorly controlled diabetes. A follow-up scan at 34 weeks and two days showed normal liquor volume (AFI = 10cm) and static abdominal circumference (on the 50th centile), while the head circumference continued to grow along the 50th centile. Her insulin requirement was also progressively being decreased as a result of repeated hypoglycaemic episodes. The findings suggested intrauterine growth restriction (IUGR) due to placental insufficiency masked by diabetes.

Delivery by caesarean section was recommended as the cervix was unfavourable on vaginal examination. With much reluctance the woman agreed to caesarean section delivery which was performed at 35 weeks and one day. A life female

infant weighing 2.1 kg was delivered. The infant was admitted to Special Care Baby Unit for 36 hours for blood glucose stabilisation before returning to mother.

Following delivery, the woman's fasting and postprandial glycaemias ranged between 4 and 9 mmol/L. Five days after delivery, a 75g oral glucose tolerance test (OGTT) was consistent with the diagnosis of type 2 diabetes with fasting plasma glucose of 4.7 mmol/L and two-hours post glucose load of 13.1 mmol/L. She remained diet-controlled on discharge but was advised that she may require insulin or oral hypoglycaemic agents (off breast feeding) very soon.

DISCUSSION

During pregnancy, various factors can precipitate and contribute to the occurrence of ketoacidosis.[4] In this instance, it may have been triggered by vomiting secondary to the urinary tract infection. A high glycosylated haemoglobin showed that the patient had significant disturbances in glucose metabolism during months prior to presentation and is suggestive of unrecognised and untreated pre-existing disease.

Caution is required with the interpretation of serial growth scans in the presence of diabetes. Polyhydramnios and macrosomia mask IUGR as relative changes in AFI and abdominal circumference will plot within normal range. A relative decrease in insulin requirement is also suggestive of placental insufficiency.

Although women with a history of gestational diabetes are at increased risk of developing type 2 diabetes, there is a high proportion who may have had unrecognised impaired glucose tolerance pre-pregnancy.[5] The increasing birth rates for older women and global epidemic in obesity further contribute to the increasing prevalence of unrecognised type 2 diabetes in women of reproductive age.[6] Therefore, timely diagnosis of gestational diabetes relies on appropriate screening of high risk groups. Unfortunately in our case there was neither evidence of a random blood glucose or risk factors at booking indicating the need for screening for diabetes.

The postpartum OGTT is consistent with the diagnosis of Type 2 diabetes according to the World Health Organisation criteria.[7] This case may represent a previously unrecognised Type 2 diabetes or an atypical form of diabetes described by Banerji, which is seen among black adults presenting with severe ketoacidosis at the onset of diabetes and a clinical course similar to that of Type 2 diabetes.[8]

In summary, diabetic ketoacidosis presenting in newly diagnosed gestational diabetes may represent a case of unrecognised Type 2 diabetes. Early recognition and prompt treatment of ketoacidosis in addition to a multidisciplinary approach is paramount to optimise outcome for the pregnant woman and her fetus.

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