

Post-partum diabetic ketoacidosis in a patient with gestational diabetes mellitus

Gestational diabetes mellitus (GDM) can be defined as glucose intolerance presenting for the first time in pregnancy.¹ Catalano *et al.*² demonstrated insulin sensitivity to reduce by up to 56% after 36 weeks' gestation. Antagonistic insulin hormones contribute to the increased resistance throughout pregnancy, perhaps explaining the increased incidence of diabetic ketoacidotic (DKA) emergencies in the second and third trimesters of pregnancy.^{3,4}

Although there is a range of data linking GDM to post-partum risk of type 2 diabetes mellitus (T2DM),^{2,5} the incidence of DKA early post-partum and the development of instantaneous type 1 diabetes (T1DM) is limited, therefore paramount importance should be given to recognising this possibility.

Case report

A 38-year-old white European woman was screened and diagnosed with GDM at 28 weeks' gestation. She was screened based on her age as a risk factor. Her oral glucose tolerance test (OGTT) was taken according to the WHO guidelines with a 75g glucose load. The fasting blood glucose was 5.5mmol/L and her 2-hour OGTT result was 8.0mmol/L.

Although capillary blood glucose (CBG) was within target range, in view of the macrosomia seen in the fetal ultrasound at 29 weeks (95th centile), the patient was started on Insulatard 4 units subcutaneously once daily and Novorapid 3 units twice daily. An elective caesarean section was planned at 37 weeks' gestation. Corticosteroids for fetal lung maturity were given according to recent guidelines on antenatal corticosteroids,⁶ i.e. corticosteroids should be given to all women for whom an elective caesarean section is planned prior to 38 weeks' gestation.

Insulin was stopped on the day of caesarean section as her insulin requirement was small. CBG was within target and HbA_{1c} was 5.6% (38mmol/mol). Postoperatively, her

CBG remained normal (range 4.5–7.0mmol) and both mother and child were well at discharge two days after caesarean section. The patient stopped monitoring after discharge.

Subsequently, the patient was admitted two days after discharge with a history of worsening nausea, vomiting and dyspnoea. On clinical examination, she was found to be drowsy, confused, dehydrated and had Kussmaul's respiration. Her blood pressure was 156/87mmHg, oxygen saturation was 98% and pulse rate 110 beats/min and she was febrile. Her plasma blood glucose level was 34mmol/L and urine analysis revealed significant ketonuria and glucosuria.

Initial investigation showed a white cell count of 22×10^9 /L and C-reactive protein of 132mg/L, indicating sepsis which was treated with intravenous (IV) antibiotics. Arterial blood gases identified a pH of 6.8 and HCO₃ of 5.0mmol/L indicating metabolic acidosis. Based on the clinical presentation and results of the investigations, the patient was also treated for DKA with IV insulin and IV fluids according to the local protocol.

The patient was discharged five days later and restarted on Novorapid three times a day and Insulatard twice daily. She was followed up by the diabetes outpatient team.

Due to her presentation with DKA and the worsening of HbA_{1c} from 5.6% (38mmol/mol) prior to delivery to 7.2% (55mmol/mol) on presentation two days after discharge, further investigations were warranted. Her anti-glutamic decarboxylase (anti-GAD) antibodies were positive, supporting the diagnosis of T1DM.

Discussion

The prevalence of GDM increases with a variety of factors including ethnicity, age, parity, obesity, weight gain and a previous history of GDM. A meta-analysis reported that women with a previous history of GDM have a 7.4-fold greater chance of developing T2DM compared to

those women who remained normoglycaemic throughout pregnancy.⁷

There are few studies which have found an association between GDM, T1DM and DKA in pregnancy.^{3,7–9} Many of these studies have been able to identify the precipitating cause of DKA. Montoro *et al.*⁸ found the main precipitating factor for the development of DKA was stopping insulin therapy. Other identified risk factors include infection and corticosteroid administration for fetal lung maturity.^{3,9} However, overall there is little supporting evidence in the literature for the development of DKA immediately post-partum in a patient with GDM.¹⁰ Inagaki *et al.*¹¹ reported a case of a woman who developed DKA and T1DM one day post-partum. However, this case differs from ours as the patient was normoglycaemic throughout pregnancy. A significant number of patients develop T1DM a few months after pregnancy; Fuchtenbusch *et al.*¹² reported the risk of T1DM was 3% by nine months post-partum and 7% two years post-partum; however, our patient developed T1DM within a week of delivery.

This case emphasises how rapidly DKA may develop and that it may present acutely. Recognition of the condition and immediate and effective management will help achieve optimum glycaemic control and reduce the potential for diabetic emergencies.

In conclusion, our case report highlights that T1DM can occur soon after delivery in subjects treated for GDM, and should be considered even if T2DM is the more likely.

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Declaration of interests

There are no conflicts of interest declared.

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