A 20-year-old woman, primigravida at 32 weeks and three days, attended her local maternity unit for a routine joint diabetes–antenatal check-up. Apart from treatment for a UTI 12 days earlier, the patient had been well and fetal movements had been normal. She was using a basal bolus insulin regimen for type 1 diabetes diagnosed at the age of 14; glycaemic control in pregnancy was suboptimal as indicated by HbA1c of 69mmol/mol; however, the capillary blood glucose (CBG) readings that she presented the diabetes team with, in the joint clinic, indicated satisfactory control.

Examination demonstrated mild bilateral ankle oedema but nil else of note, and the fetal heart rate was between 130 and 150bpm. Routine cardiotocography (CTG) monitoring to assess fetal well-being was commenced. This was initially reassuring; however, after 5 minutes, showed deep deceleration of the fetal heart rate (Figure 1), down to 85bpm, lasting more than 60 seconds. Fetal heart rate then returned to baseline but with less than expected physiological variability throughout the rest of the monitoring. With these signs of fetal distress, CBG monitoring every 30 minutes was commenced. The patient’s raised CBG was confirmed with a laboratory venous glucose of 16.2mmol/L, and urine dipstick positive for 3+ ketones and 2+ glucose. Further tests revealed venous bicarbonate of 14.6mmol/L, pH of 7.34, serum ketones of 3.4mmol/L.

On advice from the diabetes team, the obstetricians started treatment for diabetic ketoacidosis (DKA) with intravenous fluids and a fixed rate intravenous insulin infusion. She was reviewed by the diabetes specialist inpatient team and, on further questioning, admitted to missing her insulin dose, to mask her non-compliance. She was reviewed by the diabetes specialist inpatient team and, on further questioning, admitted to missing her insulin dose, to mask her non-compliance. On admission, blood ketones were 0.1mmol/L, and bicarbonate was 25mmol/L. Repeat CTG also showed marked improvement, with good variability, no decelerations, and lots of fetal movement (Figure 2).

**Discussion**

Diabetic ketoacidosis is a well-recognised medical emergency, and during pregnancy it is potentially fatal for the mother, with estimated maternal mortality of between 4% and 15%. Even in cases of maternal DKA where the mother makes a full recovery, poor outcomes for the fetus can still include stillbirth, preterm birth, and neonatal intensive care unit admissions.

A recent retrospective cohort study examining pregnancies complicated by DKA showed that poor patient compliance with the prescribed insulin had been a factor in over half of the cases. Poor compliance may be due to one, or a combination, of the following: maternal stress; reduced awareness of the maternal–fetal benefits of good diet and glycaemic control; non-engagement with the diabetes team through pre-pregnancy and pregnancy; and even an element of self-harm in purposefully neglecting diabetes control.

This case not only illustrates the importance of ensuring insulin compliance to avoid maternal DKA, but also highlights the link between the physical state of fetal health and the maternal metabolic milieu. In this patient, poor insulin compliance led to omission of the patient’s long-acting insulin, and thus to DKA. Fortunately, the observation of the abnormal CTG recording noted by the obstetrics team led to early recognition and treatment, despite the absence of symptoms in the patient. This interdisciplinary communication between different members of the health care team led to a favourable outcome for the mother and baby, and serves to highlight the importance of a collaborative approach between all members of the multidisciplinary team, including midwives, obstetricians and the diabetes team, when caring for pregnant patients with diabetes.

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**References**