was no family history of diabetes. She was on no medication and took no alcoholic beverages. On admission, she was hyperventilating, afebrile, and dehydrated and weighed 76 kg at a height of 160 cm (BMI before pregnancy was 26.9 kg/m^2); her pulse was 120/min, and her blood pressure was 100/50 mmHg. Physical examination was otherwise normal for the gestational age. The pelvic ultrasound was normal. Urinalysis revealed 4+ ketone bodies and glucose, as well as 4 leukocytes per field. Urine and cervical smear cultures were negative. Plasma glucose was 23.7 mmol/l, ketone bodies 2+, lactate 1.9 mmol/l, Na 130 mmol/l, K 4.8 mmol/l, urea 4.8 mmol/l, creatinine 88 μmol/l, HCO_3 10.7 mmol/l, hemoglobin 12.4 g/dl, and leukocytes 6,200/μl; arterial pH was 7.29, P_{CO_2} 3.03 kPa, and P_{O_2} 14.8 kPa. HbA_{1c} was 8.5%. Islet cell antibodies (ICA) were negative. The diagnosis of DKA was established. Routine therapy for DKA with intravenous normal saline, potassium, and insulin corrected the metabolic abnormalities. She was treated subsequently with daily subcutaneous insulin until delivery (37 2/7 weeks of gestation). Spontaneously, she gave birth to a macrosomic boy (4,060 g) without any complication. Immediately after delivery, fasting and postprandial glycemias returned to normal levels. At 9 months postpartum, ICA and GAD antibodies were negative. An oral glucose tolerance test (OGTT) (75 g) was compatible with glucose intolerance from fasting and 2-h plasma glucose levels of 4.6 and 8.1 mmol/l, respectively (5). Plasma C-peptide rose from 1.2 to 4.5 nmol/l during the OGTT.

To our knowledge, this is the first case of DKA during gestation without a clearly identified event predisposing to ketoacidosis. Negative urine culture excluded a suspected urinary tract infection. The O'Sullivan test performed 1 week before DKA could have precipitated the event, although this has not been previously reported in the literature. Banerji (6) described in 1994 a unique form of diabetes among black adults with severe DKA at onset of diabetes and a clinical course resembling that of NIDDM (residual C-peptide secretion capacity, GAD and ICA antibodies negative). Our patient might fit into this category because she is of African ethnicity, overweight, and insulin-resistant. We conclude that DKA, although rare, may complicate GDM without clearly identifiable precipitating factors.

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Erratum

Yosipovitch G, Hodak E, Vardi P, Shraga I, Karp M, David M, Sprecher E: The prevalence of cutaneous manifestations in IDDM patients and their association with diabetes risk factors and microvascular complications. *Diabetes Care* 21:506–509, 1998

An incorrect spelling of Elliot Sprecher's name was published in the above article. Also, Pnina Vardi and Moshe Karp each hold an MD degree.