Managing diabetic ketoacidosis in pregnancy

Sir,

Diabetic ketoacidosis (DKA) is a potentially life-threatening condition in pregnancy,[1] affecting 0.5-3% of diabetic pregnancies.[2] We describe a woman who developed DKA due to insulin pump malfunction.

A 35-year-old nulliparous diabetic, usually well-managed with a subcutaneous insulin pump, presented at 33 weeks gestation with malaise, vomiting, Kussmaul breathing and uterine contractions. Vital signs were, blood pressure 140/70 mmHg, heart rate 110 beats/min, respiratory rate 25 breaths/min and temperature 37°C. Laboratory tests were abnormal [Table 1]. The fetal heart trace showed poor variability, with late decelerations. In the intensive care unit, she received intravenous 0.9% normal saline (2 L over 3 h), then plasmalyte solution at 250 ml/h); insulin 10 u/h; and intravenous potassium. Her clinical and metabolic condition improved over 24 h [Table 1] and both contractions and late decelerations resolved. She was later discharged with a new subcutaneous insulin pump and was delivered uneventfully by elective cesarean section at 37 weeks.

Pregnancy constitutes a state of insulin resistance, accelerated starvation and respiratory alkalosis with compensatory renal bicarbonate excretion, predisposing to DKA.[2] Increased insulin resistance and/or inadequate insulin[3] may lead to hormonal-induced release of alternative energy substrates,[1] with uncontrolled hyperglycemia, dehydration, loss of electrolytes (osmotic diuresis), ketosis and metabolic acidosis.[2] The physiological response is a self-perpetuating chain of events, involving increased respiratory rate and depth (Kussmaul respiration) and compensatory low serum bicarbonate, producing an abnormal high anion gap[1] [Figures 1 and 2].

Fetal distress follows compromised uteroplacental perfusion (maternal volume depletion, acidemia and increased catecholamines),[3] and hypoxemia (decreased 2,3-diphosphoglycerate secondary to maternal hypophosphatemia).[4] Fetal hyperglycemia depletes the intravascular volume and hyperinsulinemia increases oxygen demand, depresses myocardial contractility, and combined with hypokalemia, contributes to fatal arrhythmias.[1] These changes are reversible if maternal metabolic stability is achieved.[1,2,4]

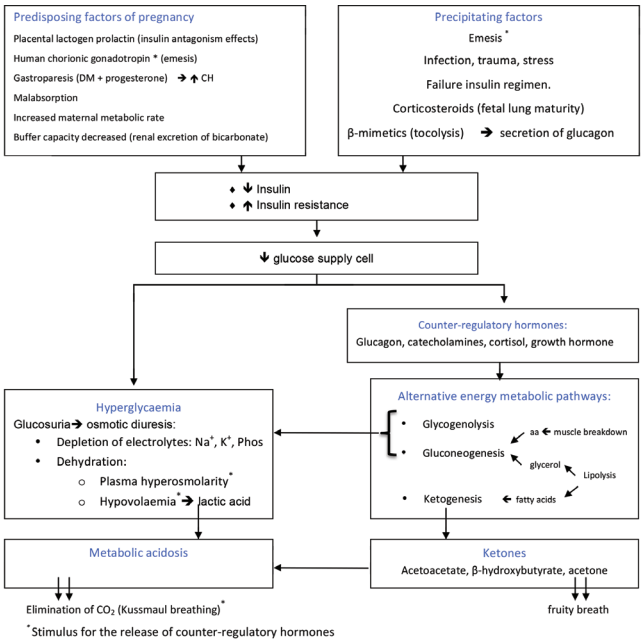

Figure 1: Pathophysiology of diabetic ketoacidosis during pregnancy. (DM: Diabetes mellitus; CH: Carbohydrates; Phos: Phosphates; aa: Amino acids)

Table 1: Sequential blood results for 24 h following admission

Hours	0	2	3	4	5	6	7	9	11	13	16	19	24
pH	7.13	7.14	7.18	7.24	7.29	7.26	7.33	7.32	7.35	7.36	7.33	7.36	7.38
$PaCO_2$ (mmHg)	20	20	20	23.6	24.9	30	25	27	26	25.5	28	29.6	29
Bicarbonate (mmol/L)	6.6	7	7.4	9.9	11	13	13	13.8	14	14	14.8	15.6	16
Base excess (mmol/L)	−21	−19	−19	−15	−13	−12	−11	−10	−9.8	−9.4	−9.4	−8	−8
Glucose (mg/dL)	383	334	298	223	252	287	236	157	143	129	90	82	99
Lactic acid (mmol/L)	4	3.9	3.2	2.5	2.4	1.8	1.6	1.4	1.1	1.1	1	0.9	1.1
Hematocrit (%)	43.7	43	39.1	38.5	38.2	36.4	34	33.9	34.3	34.5	33.7	34.8	35.1
Sodium (mmol/L)	132	135	136	137	135	135	137	135	134	139	137	133	134
Potassium (mmol/L)	5.4	3.9	3.8	4.3	4.3	4.1	3.9	3.2	3.3	3.4	3.8	4	4
Creatinine (mg/dL)	1.23			1.46			1.39			0.76			0.69

Maternal treatment must be aggressive, with reassessment of biochemical parameters 1-2 hours [Figure 2]. Intravascular volume restoration and intravenous insulin are of the highest priority to improve systemic perfusion and peripheral insulin release. Isotonic saline is mandated initially because hypotonic solutions decrease plasma osmolality (risking brain edema),[2] whereas subsequently balanced crystalloid solutions prevent iatrogenic hyperchloremic metabolic acidosis and hypernatremia.[1] Insulin corrects hyperglycemia, inhibits lipolysis and ketogenesis, suppresses hepatic glucose production and contributes to ketone body degradation.[1] Correction should be gradual, at ≤60-75 mg/dl/h, as rapidly changing serum osmolality can precipitate cerebral edema or central pontine myelinolysis.[4] Paradoxical worsening of ketoacidemia is explained by the preferential reduction of β-hydroxybutyric acid (ideally measured directly) compared with acetoacetate, which is converted to acetone, thus increasing ketone levels.[5]

Initially reduced the cellular uptake of potassium, compounded by protein degradation, predisposes to elevated serum levels, despite low total body content. Once intravenous fluid and insulin therapy are established, serum potassium may fall markedly, intravenous replacement being recommended once the concentration is below 5 mEq/L.[4]

Sodium bicarbonate does not improve outcomes and may cause harm from hypokalemia, hypernatremia, metabolic alkalosis, decreasing cerebrospinal fluid pH and compromised fetal oxygenation by left-shift of the oxygen-hemoglobin dissociation curve.[4]

Diabetic ketoacidosis in pregnancy is a medical and obstetrical emergency requiring coordinated, early aggressive treatment on a specialized unit.

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Conflicts of interest
There are no conflicts of interest.

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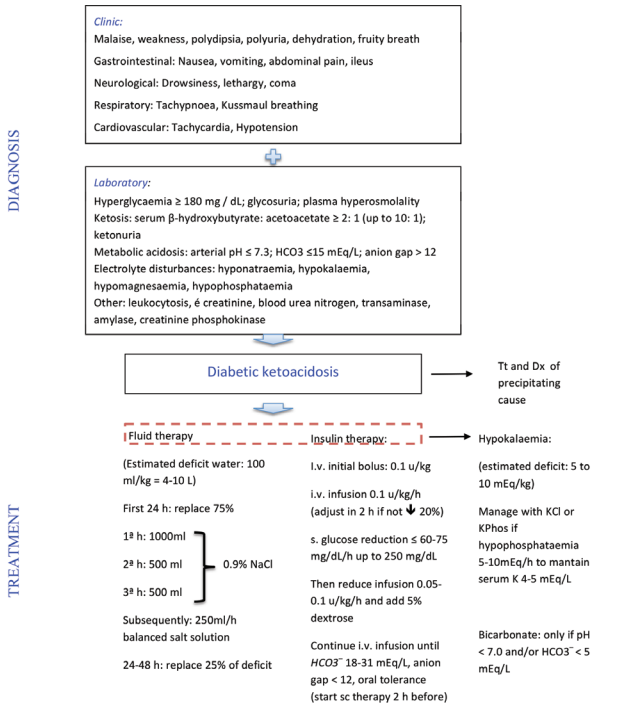

Figure 2: Algorithm for diagnosis and treatment of diabetic ketoacidosis in pregnancy. (Tt: Treatment; Dx: Diagnosis; sc: Subcutaneous)

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