Learn simply

Diabetic Ketoacidosis

Passion profession same

- Identify women at risk of developing DKA[2]
- Education to prevent DKA[3]

Confirm the diagnosis[4]

- Perform a detailed history and physical examination
- Send the following diagnostic tests: glucose, CBC, electrolytes, arterial blood gas (ABG), urinalysis, and serum and urinary ketones

Unable to confirm the diagnosis[5]

- Consider other diagnoses[6]
- Confirm gestational age
- Document fetal wellbeing

Manage as an outpatient and continue strict glycemic control

Diagnosis of DKA is confirmed[5]

- Admit to hospital
- Confirm gestational age
- Document fetal wellbeing
- Exclude infection as a cause of DKA: check blood cultures, urine culture, and a CXR if indicated

Institute treatment immediately[7]

- **Manage in ICU setting** with q 15 min maternal vital signs, EKG, facemask oxygen supplementation at 4–6 L/min
- Continuous fetal monitoring if >24 weeks' gestation

Treat hyperglycemia

- **10 units regular insulin iv push** followed by infusion of 6 units per hour (0.1 units/kg per hour) in normal saline (NS)[8]
- Check serum glucose hourly
- Aim to decrease serum glucose by 50–60 mg/dL each hour
- Stop insulin for 1 hour if serum glucose <80 mg/dL

Treat volume deficit

- Average water deficit is 10% of total bodyweight
- **Replace half of the 4–5 L fluid deficit within the first 5 hours** (~1 L per hour)
- Give NS; change to ½NS if sodium >155 mEq/L; add 5% dextrose once serum glucose <250 mg/dL

Treat electrolyte imbalance

- Add 10–40 mEq KCl per liter of iv fluid
- Check serum potassium levels hourly
- Maintain potassium levels at 4–5 mEq/L
- Stop KCl infusion if levels are >5.5 mEq/L or if there is oliguria

Treat acidosis

- *If pH <7.0, consider bicarbonate* (2 amps [88 mEq] $NaHCO_3$ in a 100 mL NS given iv over 45–60 min)
- Check acid–base status every 30 min if the pH remains <7.0[9]

Treat infection

- *Consider administering broad-spectrum iv antibiotics,* if underlying infection is suspected as the cause of DKA

Once the patient is stable, continue close observation[10]

Educate to prevent recurrent DKA

1. Diabetic ketoacidosis (DKA) results from a relative or absolute deficiency of circulating insulin in the setting of excessive glucose counter-regulatory (anti-insulin) hormones (such as catecholamines, growth hormone, cortisol, and glucagon). Insulin is an anabolic hormone that drives glucose into cells. Insulin deficiency results in a fundamental paradox: although there is an adequate supply of glucose, the body believes that it is starving and begins to make ketones for use by the vital organs (heart and brain). This leads to ketoacidosis in the setting of hyperglycemia.

2. Diabetic ketoacidosis develops in 2–10% of all pregnancies complicated by pregestational diabetes. It is extremely rare in gestational diabetes (<<1%), and effectively absent in nondiabetic women. Risk factors for the development of DKA include undiagnosed pregestational diabetes, pregnancy, emesis, noncompliance, infection, β-agonist therapy, and (perhaps) antepartum corticosteroid therapy.

3. Diabetic ketoacidosis can be effectively prevented by intensive diabetic education, rigorous glycemic control, and early identification and treatment of infection.

1. A high clinical index of suspicion is necessary to make the diagnosis of DKA. Any pregnant woman with pregestational diabetes who complains of nausea, vomiting, polydipsia, polyuria, abdominal pain, and/or decreased caloric intake should be evaluated to exclude ketosis. Physical examination may demonstrate dehydration, poor tissue turgor, tachycardia, hypotension, a fruity smell (acetone) on breath, and clinical evidence of acidosis (fatigue, hyperventilation, and Kussmaul breathing or coma).
2. The following five criteria are typically used for the diagnosis of DKA:

    1. Plasma glucose >250 mg/dL (although normal or near-normal plasma glucose levels are not sufficient to preclude DKA; indeed, up to 40% of pregnant diabetic women with DKA have plasma glucose levels on presentation of <200 mg/dL)
    2. pH ≤7.30
    3. Plasma bicarbonate ≤15 mEq/L
    4. Anion gap (calculated as $Na^+ - [Cl^- + HCO_3^-]$) >12 mEq/L
    5. Osmolality (calculated as 2 x $[Na^+ + K^+]$ + [glucose/18]) >280 mOsm/kg

3. The differential diagnosis of altered mental status in the setting of DKA includes hyperglycemic coma in women with pregestational diabetes, preeclampsia/eclampsia, seizure, drug overdose (especially alcohol), encephalopathy, uremia, infection, and psychosis.

1. Diabetic ketoacidosis is associated with a high maternal (5%) and perinatal mortality (35–50%). Other perinatal complications include preterm birth and newborn encephalopathy.

2. Prognosis depends in large part on early diagnosis and rapid and effective inpatient treatment. The primary objectives of therapy include correction of volume deficit, hyperglycemia, electrolyte imbalance, acidosis, and treatment of the precipitating cause (such as infection). Fetuses die of acidosis and not high glucose levels. As such, the immediate goal of treatment is reversal of ketoacidosis, not euglycemia.

3. The half-life of IV insulin is 2–4 min. DKA can recur in the absence of exogenous insulin. Subcutaneous insulin should therefore be restarted once the patient is eating.
4. If the acidosis persists despite initial treatment, consider inadequate insulin administration, sepsis, or hypophosphatemia.

1. Once stable, it is important to:

(i) follow fingerstick blood glucose hourly;
(ii) check serum electrolytes and arterial blood gas (ABG) q 2–4 hourly, as indicated;
(iii) check BUN/creatinine and urinary ketones q 4 hourly;
(iv) catheterize patient if unconscious or not passing urine;
(v) decompress stomach if unconscious; and
(vi) undertake continuous fetal surveillance and delivery, if indicated.