DKA in pregnancy

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Background

- 25 year old G6 P4104
- 18 1/7 weeks’ gestation
Initial Presentation

- 4th triage visit with complaint of nausea & vomiting
- Anti-emetics not working
- Recently treated for UTI, but delayed in getting prescription
Past Medical History

- Type I DM diagnosed age 21
- Initial diagnosis of DKA in pregnancy
- Multiple admissions with DKA
- Poor compliance
1. 1997 SVD
2. 1999 primary LTCS secondary to failure to progress
3. 01/2001 VBAC (normal 1 hr screen for gestational diabetes)
4. 12/2001 DKA diagnosed at 23 weeks' gestation VBAC
5. 2004 DKA at 33 weeks and FDIU at 36 weeks with VBAC
• Family Hx: None
• Social Hx: No toxic habits
• Medications:
  o Lantus 27 units qhs
  o NovoLog 13 units qac
  o Macrobid 100 mg daily (day 2 of her course)
• Prenatal Labs
  o MSAFP within normal limits
  o 24-hour urine protein 154 mg
  o Hemoglobin A1C of 5.2
Physical Examination

- Patient was comfortable and in no distress
- VS: T 37.1 P 96 BP 124/68 R 22
- Positive fetal heart tones
- Weight 75 kg
- Serum glucose 163 mg/dL
- Urine dip +glucose
Assessment/Plan

- Hyperemesis
- Ondansetron and IV normal saline
- Blood sample unable to be drawn after multiple attempts
- The patient felt much better after two hours of hydration and was discharged home
Second Presentation

- Returns to triage same day
- Complaints of nausea and vomiting, severe fatigue, and a headache
- On further questioning, reports increase in urinary frequency and general malaise over the past few days
- Because of her nausea and vomiting this morning did not take her insulin
Physical Examination

- Dyspnic, alert and oriented x 3
- VS: T 37.4   P 118   BP 102/55   R 41
- Oxygen saturation 99%
- Positive fetal heart tones
- HEENT: Oral mucous membranes somewhat dry, no neck lymphadenopathy
- CVE: tachycardia with a regular rhythm and no murmurs, rubs, or gallops
Physical Examination

- Lungs: clear to auscultation bilaterally
- Back: no CVA tenderness
- Abdomen: soft, nontender, nondistended, uterus nontender
- Extremities: no edema
- Serum glucose 413 mg/dL
Labs

- CBC: Wbc 23  Hct 32  Plt 358  bands 13 (↑)
- Electrolytes:  Na 135  K 3.9  Cl 109  HCO3 10
  BUN 10  Cr .6  GLU 303  Phos 1.1
- Anion gap 19
- ABG: pH 7.18  PaCO2 18  HCO3= 7; PaO2= 105
- Base excess -20
- Urinalysis: +Leuk, +glucose, +ketones, -nitrite
- EKG: sinus tachycardia with a few nonspecific ST and T wave changes.
Learning Objectives

- Describe the major metabolic changes of DKA
- Describe the major causative factors for DKA
- List clinical signs and symptoms of DKA
- Choose laboratory and diagnostic studies confirming the diagnosis
- Discuss medical management of DKA
- Discuss potential complications and prognosis to both patient and fetus
Diagnosis → DKA
Hospital Course

- IV fluid 1000mL NS for one hour
- Continuous IVF half NS at 250cc
- 10 unit bolus of insulin continued at 5 units per hour as a drip
- Intravenous antibiotic
- Serum glucose checked hourly
- Dextrose added to IVF when serum glucose 260 mg/dL
Hospital Course

- Hydration was continued until acidosis resolved
- Serum phosphate and potassium were replaced
- Started on a diabetic diet and resumed subcutaneous insulin administration
- Ultrasound performed showing a live fetus appropriately grown
- Presumed cause of DKA from an untreated UTI
- Discharged home hospital day #4 in stable condition
Diabetic Ketoacidosis

- Incidence decreasing
  - 1-3% in during pregnancy
- Most often occurs in type I DM
  - Case reports on gestational DM and type II DM
- Mortality <5%
Pathophysiology of DKA

- Lack of insulin or insulin utilization at cellular level
- Derangement of carbohydrate, protein and lipid metabolism
- Increase in counterregulatory hormones (glucagon, catecholamines, cortisol, growth hormone)
Pathophysiology of DKA

- Hyperglycemia
- Metabolic acidosis
- Elevated plasma ketones
- Dehydration
INSULIN DEFICIENCY

↑ COUNTERREGULATORY HORMONES

↑ LIPOLYSIS

↓ GLUCOSE UTILIZATION

↑ PROTEOLYSIS

↑ GLYCOGENOLYSIS

↑ GLUCONEOGENESIS

↑ FREE FATTY ACIDS

↑ KETOGENESIS

↑ SERUM KETOACIDS

METABOLIC ACIDOSIS

HYPERGLYCEMIA

GLYCOSURIA (OSMOTIC DIURESIS)

ELECTROLYTE IMBALANCE

DEHYDRATION (HYPOVOLEMIA)
Pregnancy Physiology Predisposing DKA

- Hyperventilation (increase in minute ventilation and tidal volume)
- Mild respiratory alkalosis
- Increase in urinary excretion of bicarbonate
- Emesis (common in first trimester) induces dehydration
- State of increased insulin resistance
Decrease insulin sensitivity (56% at 36 weeks gestation)

Diabetogenic hormones increase (human placental lactogen, progesterone, and cortisol)

State of accelerated starvation (metabolism is modified to breakdown fats over carbohydrates)
Precipitating Factors of DKA

- Infection
- Stress
- Emesis
- Poor compliance
- Undiagnosed diabetes
Diagnosis of DKA

- Hyperglycemia (>250 mg/dL)
- Acidosis (arterial pH < 7.3)
- Anion gap (>12 mEq/L)
- Increased base deficit (>4 mEq/L)
- Ketonemia (≥1:2 dilution)
- Some patients (36 percent in one series) may have glucose levels less than 200 mg/dL
Fetal effects of DKA

- Perinatal mortality has dropped from about 35% to 10
- Hypoxemia (volume depletion and acidosis may lead to decreased uterine blood flow)
- Metabolic acidosis (glucose and ketones readily cross the placenta)
Fetal effects of DKA

- Fetal heart rate pattern
  - Absence of baseline heart rate variability
  - Persistent late decelerations
  - Non-reassuring biophysical profile

- Prompt and aggressive treatment of maternal condition has been shown to improve the fetal status
Emergency cesarean delivery could worsen the maternal condition and should be avoided.

After having corrected the maternal metabolic condition, a non-reassuring fetal heart rate may require intervention.
Treatment Goals

- Volume replacement (saline)
- Stop ketogenesis (insulin)
- Electrolyte replacement
- Management of acid-base disturbance
- Identify and treat underlying cause
Fluids

- 1000mL 0.9% NaCl over one hour
- 500mL 0.45% NaCl next four to six hours (Adjust fluid according to electrolytes)
- 250mL 0.45% NaCl
- Add 5% dextrose when serum glucose reaches 250-300 mg/dL
- Correct 75% of estimated fluid deficit over first 24 hours
- Continue IV fluid until acidosis is corrected (base excess -2 or less)
Insulin

- 0.1-0.2 units/kg regular insulin IV bolus followed by continuous infusion at a rate of 5-10 units/hour
- Goal of therapy is a reduction of glucose by about 75 mg/dL/hr
- If glucose does not decrease by 20% within first 2 hours, may increase insulin rate
- May decrease to 2 units/hour when serum glucose is 250-300 mg/dL and HCO3 ≥ 18mEq/L
Potassium

- Maintain between 4 and 5 mEq/L
- If normal value, may add 20 mEq/L to fluid to maintain level
- Serum potassium initially may be normal or high because of a shift from the intracellular to extracellular space due to hyperosmolality and acidosis
- Potassium should not routinely be given in initial fluids because in a volume contracted state with no insulin present it can rise quickly and cause arrhythmias
Treatment

- Search for precipitating cause
- Check serum glucose hourly
- Check arterial blood gas, electrolytes, and anion gap every 2-4 hours
- Careful monitoring of vital signs, urine output, and
- Fetal monitoring if over 24 weeks’ gestation


