DIABETIC KETOACIDOSIS (DKA)

(Last updated 07/23/2019; Reviewed by: Kirtivardhan Vashistha, MBBS)

PRESENTING COMPLAINT: Hyperventilation, Abdominal pain, Altered mental status, fruity odor

FINDINGS

- **A** Check airway
- **B** ↑ RR, fruity odor on breath
- **C** ↑ HR, may have weak pulses,
- **D** Variable altered (V,P,U,D) *
- **E** Decreased skin turgor, dry axillae, dry mucosa
- **L<sub>PC</sub>** ↑ Glucose (usually >250 mg/dL); ↓ Arterial pH < 7.3; ↑ Anion gap > 12, Ketonuria;
  ↑ Serum B-hydroxybutyrate > 3 mmol/L; ↓ Serum bicarbonate < 18 mEq/L
  Hyperkalemia, Hyponatremia, ↑ Posm; ↑ BUN/Cr
- **U<sub>PC</sub>** Not pertinent

*V (verbal), P (pain), U (unconsciousness), D (delirious)  

U<sub>PC</sub> (point of care ultrasound)  L<sub>PC</sub> (point of care labs)

OTHER HISTORY

- Abdominal pain, nausea/vomiting, decreased skin turgor, dry mucosa, weight loss, seizure,  
  stupor/coma, polyuria, polydipsia

DIFFERENTIAL DIAGNOSIS

- Hyperosmolar hyperglycemic state, alcoholic or fasting ketoacidosis, lactic acidosis, ingestion of 
  drugs (aspirin, methanol, ethylene glycol), advanced chronic kidney disease, acute abdomen, sepsis

OTHER INVESTIGATIONS

- Labs
  - Electrolytes (potassium and anion gap), arterial/venous pH, plasma osmolality (high 
    normal/mildly elevated), lactate, urine or serum ketone bodies (e.g beta-hydroxybutyrate] 
    high), blood count, urinalysis, sodium adjusted for degree of hyperglycemia
    - Serum sodium concentration should fall by about 1.6 mEq/L for each 100 mg/100 mL [5.5 mmol/L] increase in glucose concentration
  - Additional tests: Blood cultures and chest x-ray (If suspected infection), electrocardiogram and 
    cardiac enzymes (If cardiac ischemia suspected), blood ethanol levels, drug screen
  - Monitoring
Fluid/volume status, point of care glucose q1h, electrolytes (sodium, potassium, phosphorus, anion gap), BUN, creatinine, q2-4h (until anion gap has closed)

THERAPEUTIC INTERVENTIONS

- Fluid therapy
  - Low corrected sodium
    - 1 L normal saline bolus followed by infusion: 4-14mL/kg lean body weight per hour (250-500mL/h), according to hydration status
    - If patient is in shock, consider rapid 2-4L bolus in first hour
  - Normal/elevated corrected sodium
    - 1 liter 0.45% NaCl bolus followed by infusion: 4-14mL/kg lean body weight per hour (250-500mL/h), according to hydration status
  - When Glucose <250 mg/dL
    - Initiate 5% dextrose in 0.45% NaCl at 150-200 ml/hour
      - Dextrose infusion rate may need to be increased if serum glucose is less than 150 mg/dL to allow insulin infusion to continue
    - Insulin infusion should NOT be stopped for hypoglycemia
  - Consider risk of fluid overload if renal/cardiac failure, esp. if age > 65yrs or hypoxemia

- Insulin therapy
  - Regular insulin or insulin analogs
    - Patient should remain nothing by mouth while on insulin infusion
  - If serum potassium is <3.3 mEq/L, administer potassium replacement until level is > 3.3 mEq/L prior to/concurrent with starting insulin infusion
    - Initially
      - Continuous IV infusion of 0.14U/kg/h regular insulin
        - Alternatively, IV bolus of 0.1U/kg followed by infusion of 0.1U/kg/h
        - If glucose decreases by less than 50-70mg/dl (2.8-3.9mmol/L) from initial value within the first hour, double infusion rate each hour until glucose decline becomes steady
      - Once serum glucose reaches 200mg/dl (11.1mmol/L): decrease infusion rate to 0.02-0.05 U/kg/h to maintain glucose level between 150-200 mg/dl until anion gap closes
      - Once anion gap is <12 mEq/l, blood glucose <200mg/dl, and patient is able to eat:
• Administer subcutaneous, long-acting insulin: home insulin regimen or 50% of 24 hour insulin infusion requirement or 0.2 units/kg/day
• Stop insulin and dextrose infusion 4 hours after subcutaneous insulin administration

• Hypokalemia
  o If normal renal function, start supplementation with IV potassium as soon as potassium < 5 mEq/L
    ▪ < 3.3 mEq/L: hold insulin therapy and give 20-40 mEq/L until level > 3.3 mEq/L
    ▪ 3.3-5.3 mEq/L: add 20-30 mEq/L to each liter of crystalloid to maintain serum K 4-5 mEq/L
    ▪ > 5.3 mEq/L: hold supplemental K and continue checking level q2 hours
  o Consider decreased renal function

• Bicarbonate therapy: Consider if life-threatening hyperkalemia

• Empiric antibiotics: Only if suspicion of infection; Deescalate rapidly

ONGOING TREATMENT
• Check HbA1C
• Treatment
  o Increase diet once nausea/vomiting resolved and patient is off insulin infusion
  o Consider phosphorus/magnesium repletion
  o Patient education if insulin noncompliance or new onset diabetes

CAUTIONS
• Mental and/or neurologic status alteration may be sign of cerebral edema
• Always carefully evaluate the patient for an underlying cause of DKA.
• Underlying infection is common and may be life threatening: look for an infectious source and treat with appropriate antibiotics

REFERENCES & ACKNOWLEDGEMENTS
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Kitabchi AE and al. Hyperglycemic crises: Diabetic Ketoacidosis (DKA), and hyperglycemic hyperosmolar state (HHS).