

DKA

Diagnosis

DKA vs HHS:

- DKA sugar ranges from 250 to 500 mg/dL
- HHS typically has sugars > 600 mg/dL
- Serum osmolality > 320 in HHS
- Neurologic symptoms such as stupor and coma are primarily seen in HHS, but can sometimes be seen in severe DKA.
- Little to no ketonuria/ketonemia in HHS
- Anion gap mainly in HHS

There may be focal neurological deficits and/or seizures in HHS.

In DKA, there may be nausea, vomiting, and abdominal pain. The delayed gastric emptying and/or ileus caused by acidosis and electrolyte derangement may be a cause for such symptoms.

Kussmaul respirations are indicative of acidosis since this is a compensatory mechanism. A fruity breath may be from acetone, which is one of the three ketone bodies produced.

Infection or inadequate control of DM are the main causes. Sometimes DKA is the first presentation for DM.

Laboratory and Ancillary Testing

First assess clinical stability of the patient. Ability to protect airway and mental status assessment are important assessments.

A part of the quick assessment is the following STAT orders... (these should already be ordered by ED, but always make sure these are ordered when you accept an admission)

- Basic metabolic panel
- Urine analysis
- Venous blood gases
- Beta Hydroxybutyrate level
- CBC
- CXR (if respiratory symptoms)

- Influenza panel (if in season)
- Blood cultures (if septic)

* remember that in DKA the total water deficits are 6L and for HHS 9L.

Common Laboratory Derrangements

Hypонатremia is often present due to fluid shifting from ICF to ECF. Basically the hyperglycemia causes fluid shifts from ICF to ECF. Be cautious of patients with impaired thirst mechanisms and the summer months where hot weather can cause dehydration.

For every 100 mg/dL increase in serum glucose, expect a 2mEq/L fall in serum sodium.

Hypokalemia or hyperkalemia can be present. Increased osmotic diuresis due to hyperglycemia can cause losses of potassium. Conversely, elevated potassium can be caused by ICF to ECF shifts of potassium due to lack of insulin or significant insulin resistance.

Hypophosphatemia or hyperphosphatemia is present. Dietary causes such as low phosphate intake can be present in uncontrolled DM. Conversely, hyperphosphatemia can be present in metabolic acidosis and hyperglycemia due to ICF to ECF shifts of phosphate.

Lipase and amylase are often elevated and DKA can masquerade as pancreatitis. Clinical judgement is necessary to distinguish the two.

Leukocytosis is present in many DKA or HHS patients. Hypercortisolemia and metabolic potentiators such as excess catecholamines can cause this. Think stress response. Do not forget to think about infectious etiology, WBC > 25K or bandemia with > 10%.

Acute Kidney Injury in the form of increased creatinine or reduced GFR can be present. In this case think dehydration or hypovolemia.

Hyperlipidemia can be present and increased lipolysis can serve as substrates for ketone body generation.

DKA and HHS are treated the same for the most part. It is important to think of the four hallmarks of DKA and HHS treatment which are correction of ...

- Hyperglycemia
- Electrolyte derangement
- Acidosis
- Dehydration

Use the order set in the hospital

1. Fluid Replacement

- a. In the first 2 hours, isotonic (Normal Saline) fluid boluses (1L/hr in average sized person). Be cautious and individualize care on CHF or fluid intolerant patients.
- b. In the 3rd hour, continue fluids using either normal saline or half normal saline. If sodium is below reference range, use NS at 250 to 500 mL/hr. If sodium is normal or above reference range, then half normal.

2. Insulin gtt (regular insulin)

- a. 0.1 units/kg bolus followed by 0.1 units/kg/hr.
- b. If no bolus is used, then 0.14 units/kg/hr.
- c. Nursing will adjust rate based on serial glucose measurements.

Caution - if potassium is < 3.3 , correct the hypokalemia first otherwise you will tank the patient's potassium which is dangerous.

Management and Monitoring

3. Electrolytes – there is a protocol for electrolyte correction on the hospital order set. Be sure to use this.

- a. Potassium – again replete potassium first if value is < 3.3 . May need to add KCl to replacement fluids above (20 to 40 mEq/hr) if potassium is between 3.3 and 5.5 mEq/L.
- b. Sodium – this will correct with correction of hyperglycemia.
- c. Phosphorus – Correct when < 1 . If there is a cardiac history or potential for arrhythmia, correct if < 3 . Sodium phosphate can be used.

4. Acidosis – No indication for bicarb unless the pH is ≤ 7.35 or if potassium > 6.4 mEq/L.

- a. 100 mEq sodium bicarbonate in 400 mL of water.
- b. Keep in mind that bicarb administration can cause hypokalemia or worsen it.

Monitoring includes....

- Q1h glucose checks
- Q2h metabolic panel
- Q2h VBG

Resolution of DKA and HHS

What we look for...

- Normalization of anion gap (<12)
- Absence of serum beta hydroxybutyrate level.
- Mental lucidity in HHS and plasma osmolality < 315
- Drop in glucose levels to 250 to 200 mg/dL
- Tolerance to PO diet.

CONVERSION TO SUBCUTANEOUS INSULIN

Only initiate subcutaneous insulin if the patient is able to eat and the above criteria regarding resolution of ketoacidosis is met.

HHS

1. **Overlap 2 hours of insulin gtt** when serum glucose reaches 250 to 300 mg/dL

DKA

1. **Overlap 2 hours of insulin gtt** when serum glucose reaches 200 mg/dL
2. In addition two of the following must be met
 - a. Anion gap < 12
 - b. Bicarb ≥ 15
 - c. Venous pH > 7.30

References:

1. Up to Date has the latest and best information
2. SAMC order set for DKA
3. Dr. Michael Moya's DKA Handout

