Mind the Gap: Navigating the Underground World of DKA

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Objectives

• Upon completion of this activity the learner will be able to describe the physiology of DKA

• Upon completion of this activity the learner will be able to describe the nurse’s role in treatment strategies for DKA

Back That Train Up!

• Insulin has a number of effects on glucose metabolism, including:
  – Inhibition of glycogenolysis and gluconeogenesis
    • Insulin hits the breaks on extra glucose conversion or production
  – Increased glucose transport into fat and muscle
    • Insulin takes glucose on a sweet ride into the fat and muscle
  – Increased glycolysis in fat and muscle
    • Insulin fuels the generation of ATP
  – Stimulation of glycogen synthesis
    • Insulin parks the glucose train at the station until it’s needed
So what causes DKA?

How do you know you’re on the right train?

<table>
<thead>
<tr>
<th>Diabetic Keto Acidosis</th>
<th>Hyperosmolar Hyperglycemic State</th>
</tr>
</thead>
<tbody>
<tr>
<td>Type I Diabetes</td>
<td>Usually associated with:</td>
</tr>
<tr>
<td>DKAn results from an insulin deficiency; in response the body switches to burning fatty acids and producing acidic ketone bodies</td>
<td></td>
</tr>
<tr>
<td><strong>&gt;250</strong> Plasma glucose (mg/dL)</td>
<td></td>
</tr>
<tr>
<td><strong>&lt;7.00-7.30</strong> Arterial pH</td>
<td></td>
</tr>
<tr>
<td><strong>&lt;10-18</strong> Serum Bicarbonate</td>
<td></td>
</tr>
<tr>
<td>+ Urine Ketones Small</td>
<td></td>
</tr>
<tr>
<td>+ Serum Ketones &lt; 0.6mmol/L</td>
<td></td>
</tr>
<tr>
<td>Variable Serum Osmolality</td>
<td></td>
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<tr>
<td>&gt;10-12 Anion Gap Variable</td>
<td></td>
</tr>
</tbody>
</table>

HHS results from an insulin deficiency that leads to a serum glucose that is usually higher 600 mg/dl, and a resulting high serum osmolality

- Hyperglycemia causes osmotic diuresis
- Ketones and electrolytes are lost
- Insulin deficiency
- Metabolism of triglycerides and fatty acids for energy
- Serum glycerol, free fatty acids and alanine levels rise
- Glycerol and alanine stimulate hepatic gluconeogenesis
- Free fatty acids are converted to ketones
Ticket to Trouble: Signs & Symptoms

- Hyperglycemia
- Polyuria & polydipsia
  - Dehydration
  - Hypotension
  - Tachycardia
- Nausea & vomiting
  - Electrolyte imbalances
- Abdominal pain
- Hyperventilation
  - Kussmaul Respiration
- Neurologic symptoms
  - Lethargy
  - Focal deficits
  - Obtundation
  - Seizure
  - Coma

<table>
<thead>
<tr>
<th>Mild</th>
<th>Moderate</th>
<th>Severe</th>
</tr>
</thead>
<tbody>
<tr>
<td>Arterial pH</td>
<td>7.25-7.30</td>
<td>&lt;7.00</td>
</tr>
<tr>
<td>Serum</td>
<td>10-18</td>
<td>10 to &lt;15</td>
</tr>
<tr>
<td>Bicarbonate</td>
<td>&gt;30</td>
<td>&gt;32</td>
</tr>
</tbody>
</table>

Mind the Gap: Anion Gap

- Anion: a negatively charged ion
  - Famous anions include: CL-, HCO3-, NO3-, CO3-
- Cation: a positively charged ion
  - Famous cations include: K+, NA+, Mg+, Ca+
- Serum AG = Measured cations - measured anions
- Normal = 0-15

\[ AG = NA - (CL + HCO_3) \]

BRAKE CHECK

DKA is a Clinical Emergency

The goals of treatment include:
- Restoration of plasma volume & perfusion
- Reduction in blood glucose & osmolality
- Correction of acidosis
- Replenishment of electrolytes
- Identification of precipitation factors
First Stop: Fluids

- Most patients present with a fluid deficit of 4-5 liters
- Anticipate an order to infuse 0.9% normal saline:
  - 1 liter/hr for the first 1-2 hours
  - 300-400 mL/hr thereafter
- You’ll change the fluids to 5% Dextrose later

Second Stop: Insulin

- Anticipate initiating a continuous intravenous infusion of REGULAR insulin
  - Loading dose 0.15 units/kg primes the tissue insulin receptors
  - Initiate the infusion per hospital protocol or physician order
    - The typical basal infusion is 0.1 unit/kg/hr
  - If plasma glucose doesn’t fall at least 10% in the first hour, you may need to repeat the loading dose

Delay insulin infusion if the potassium level is < 3.3 mEq/L

Insulin infusion will worsen the hypokalemia by driving potassium into the cells, triggering cardiac arrhythmias
Insulin, continued

- Insulin treatment:
  - Restores normal metabolism
  - Reduces hyperosmolality
    - Increases peripheral use of glucose
    - Decreases hepatic glucose production
  - The need for IV insulin should resolve within 24 hours

BRAKE CHECK

Avoid rapidly decreasing serum glucose

Decreases of >150 mg/dL per hour increase the potential for cerebral edema

Signs and symptoms of cerebral edema include but are not limited to: headache, decreased level of consciousness, hallucinations, and coma

Alternate Route: SQ Management

- A randomized trial of 45 patients in DKA received SQ aspart (Novolog) or IV regular insulin
  - Outcomes were identical
  - SQ insulin was given every 1-2 hours

- A meta-analysis of 4 studies (155 patients, with the above study included) supports SQ rapid-acting insulin analogues as an alternative to IV insulin in uncomplicated DKA
Third Stop: Watch those electrolytes!

- Potassium
  - K+ is usually close to normal prior to treatment because it shifts into the extracellular space
  - As treatment begins K+ shifts back into the cells
    - Hypokalemia can occur
  - K+ replacement should start ~2-3 hours into therapy
    - OR, if the K+ was <3.3 mEq/L, replacement starts before insulin is given

Fourth Stop: An Acidic Environment

- Sodium Bicarbonate
  - Slow and careful replacement is recommended when the pH is ≤7.0
    - Anticipate administration of 0.45% NS with HCO3
    - The infusion should be stopped when pH ≥7.1
  - Potential complications of bicarb replacement
    - Overcorrection can cause hypokalemia
    - Tissue anoxia can occur when acidosis is rapidly overcorrected
    - Cerebral acidosis lowering of pH in CSF

Monitoring

- Monitor:
  - Glucose hourly
  - Electrolytes, plasma osmolality, and venous pH every two to four hours
  - Cardiac rhythm
  - Mental status per unit protocol
- The patient should be NPO
Parallel Tracks: Closing the Anion Gap

- The anion gap may still be open even when the blood glucose is <250mg/dL
  - Continue the insulin infusion
  - Expect an order for dextrose-containing fluids to support blood glucose

Pulling into the Station

- Anticipate transition to SQ insulin when:
  - The patient alert & oriented, able to eat
  - The anion gap is closed
- Administer SQ insulin 2 hours prior to discontinuing the insulin infusion
  - The insulin regimen should include orders for long-acting basal insulin and short-acting bolus and prandial insulin
- DKA can cause a transient tissue insulin resistance
  - This may be seen for a few days with resultant decrease in insulin requirements

References


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