Diabetic ketoacidosis and hyperglycaemic hyperosmolar state

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Where do I come from?
You can find us on FaceBook..

DKA and HHS are life-threatening emergencies

<table>
<thead>
<tr>
<th>Diabetic Ketoacidosis (DKA)</th>
<th>Hyperglycaemic Hyperosmolar State (HHS)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Plasma glucose &gt;250 mg/dL (&gt;14 mmol/l, usually much higher)</td>
<td>Plasma glucose &gt;600 mg/dL (&gt;33 mmol/l)</td>
</tr>
<tr>
<td>Arterial pH &lt; 7.3</td>
<td>Arterial pH &gt; 7.3</td>
</tr>
<tr>
<td>Bicarbonate &lt; 15 mEq/L</td>
<td>Bicarbonate &gt; 15 mEq/L</td>
</tr>
<tr>
<td>Moderate to severe ketonuria or ketonaemia</td>
<td>Minimal ketonuria and ketonaemia</td>
</tr>
<tr>
<td>Anion gap &gt;12 mEq/L</td>
<td>Serum osmolality &gt;320 mosm/L</td>
</tr>
</tbody>
</table>

Acidosis  Dehydration
Case presentation - 1

- Woman, 39 years of age
- Referred to First Aid Dept
- Did not respond to husband trying to wake her up
- History:
  - Psychiatric complaints, possibly agorafobia?
  - Known to have diabetes, referring GP uncertain about details of treatment
- History of spouse:
  - Since 12 hours nausea, vomiting, has not eaten much, but did drink tea and water

This is a real patient

Case presentation - 2

- E1M1V1, middlewide pupils, not reacting to light
- Smells like fresh fruits, like ‘pears’
- Blood pressure 80/40mmHg, HR 80/min, rectal temperature 28.1 °C
- Heart: soft heart sounds, no murmur. Lungs: normal
- Abdomen: no bowel sounds, hypertympanic, clapotage* +
- Other: calcified nails hand and feet; several small pustulae on the body, larger abscess at the back
- After insertion of bladder catheter and gastric tube
  → bladder retention 1000cc, 1200cc of fluid in stomach

* splashing sound heard with a dilated fluid-filled stomach
Case presentation - 3

- Arterial blood gas analysis:
  - pH 6.66, pCO₂ 2.9, PO₂ 14.5, bicarbonate 3, saturation 94%.
  - sodium 138 mmol/l, potassium 2.7 mmol/l
- Other lab:
  - White blood cells 66.4, Hb 9.9, platelets 689,
  - Alk phosphatase 438, LDH 748, ASAT 42, ALAT 39
  - calcium 2.27, albumin 31, chloride 98, creatinin 72
  - CPK 131 U/l
- Conclusion: DKA with skin infections & cessation of insulin

Clinical presentation of diabetic ketoacidosis

**History**
- Thirst, polyuria
- Abdominal pain
- Nausea and/or vomiting
- Profound (muscular) weakness
- Drowsiness, confusion

**Physical Exam**
- Kussmaul respirations
- Fruity breath
- Relative hypothermia
- Tachycardia
- Supine hypotension, orthostatic drop of blood pressure
- Dry mucous membranes
- Poor skin turgor
Laboratory findings in DKA

- Severe hyperglycaemia
- Increased blood and urine ketones
- Low bicarbonate
- High anion gap
- Low arterial pH
- Low PCO₂ (respiratory compensation)

Help, my patient with diabetes is unconsciousness! What may be the cause?

- Diabetic ketoacidosis
- Hyperosmolar non-ketotic hyperglycaemia
- Hypoglycaemia
- Lactic acidosis
- Other causes, like stroke, post-ictal (after hypo?), cerebral trauma, alcohol intoxication, drug overdose, etc.
Deficiency of insulin & Excess of contra-regulatory hormones
- Glucagon, catecholamines, cortisol, growth hormone

 unchecked gluconeogenesis → Hyperglycaemia
Osmotic diuresis → Dehydration
Lipolysis, FFA breakdown, ketogenesis → Ketosis
Dissociation of ketone bodies into hydrogen ion and anions → Anion-gap metabolic acidosis
Acidosis → Negative inotropic, vasodilation, worsening hypotension

Often a precipitating event is identified (infection, lack of insulin administration)
Hyperosmolar hyperglycaemic state: pathophysiology

Unchecked gluconeogenesis → Hyperglycaemia

Osmotic diuresis → Dehydration

- Presents commonly with renal failure
- Insufficient insulin for prevention of hyperglycaemia, but sufficient insulin for suppression of lipolysis and ketogenesis
- Absence of significant acidosis
- Often identifiable precipitating event (infection, MI)
Admission clinical and biochemical profile and response to therapy of comatose vs noncomatose patients with DKA

<table>
<thead>
<tr>
<th></th>
<th>Noncomatose n=35</th>
<th>Comatose n=13</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (yr)</td>
<td>36.1 ± 3.9</td>
<td>50.2 ± 6.8</td>
<td>&lt; 0.02</td>
</tr>
<tr>
<td>Glucose (mmol/l)</td>
<td>32.1 ± 2.4</td>
<td>54.9 ± 9.7</td>
<td>&lt; 0.01</td>
</tr>
<tr>
<td>HCO₃⁻ (meq/l)</td>
<td>8.6 ± 0.7</td>
<td>6.1 ± 0.9</td>
<td>&lt; 0.02</td>
</tr>
<tr>
<td>pH</td>
<td>7.19 ± 0.25</td>
<td>7.10 ± 0.45</td>
<td>NS</td>
</tr>
<tr>
<td>Osmolality(mOsmol/kg)</td>
<td>314 ± 2</td>
<td>365 ± 15</td>
<td>&lt; 0.01</td>
</tr>
<tr>
<td>Ketones (mM)</td>
<td>13.7 ± 0.8</td>
<td>14.3 ± 1.4</td>
<td>NS</td>
</tr>
</tbody>
</table>

**Hours to Recovery**

<table>
<thead>
<tr>
<th></th>
<th>Noncomatose</th>
<th>Comatose</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Glucose ≤ 14mmol/l</td>
<td>5.2 ± 0.6</td>
<td>9.5 ± 2.5</td>
<td>&lt; 0.05</td>
</tr>
<tr>
<td>HCO₃⁻ &gt; 15meq/L</td>
<td>10.6 ± 1.7</td>
<td>12.9 ± 2.7</td>
<td>NS</td>
</tr>
<tr>
<td>pH ≥ 7.3</td>
<td>6.6 ± 1</td>
<td>10.2 ± 2.8</td>
<td>NS</td>
</tr>
<tr>
<td>Mentally alert</td>
<td>NA</td>
<td>7.8 ± 4.2</td>
<td>NA</td>
</tr>
</tbody>
</table>

Diabetic ketoacidosis (DKA) - issues for discussion

- Fluid resuscitation
- Shot of insulin at start of treatment
- Intravenous vs SC. vs IM. insulin therapy
- When to give bicarbonate
- When to give phosphate suppletion
- How to give potassium suppletion

Is there any ‘evidence’?
Is it explained by pathophysiologic reasoning?
Is it warranted, safe, or of benefit?
Diabetic ketoacidosis (DKA) - issues for discussion

- One severe complication is called:
  
  *Central pontine and extra-pontine myelinolysis (CPM/EPM)*

- Is a clinical picture which develops after too rapid restoration of hyponatremia or due to hyponatremia alone: dysarthria, dysphagia ('bulbar paralysis'), tetraparesis/plegia

- MRI shows abnormal signal intensity in the pons, deeper layers of the cerebral cortex, and adjacent white matter

Diabetic ketoacidosis (DKA) - treatment: insulin administration

- Shot of insulin at start of treatment
- Intravenous vs SC. vs IM. insulin therapy
Diabetic ketoacidosis (DKA) - treatment: insulin administration

- High-dose group (90 U initially) (50 U/hr SC)
  - Plasma insulin, μU/mL
  - Glucose
  - Hours after insulin injection

- Low-dose group (0.22 U/kg B. Wt.) (5 U/hr IM)
  - Plasma glucose, mg/100 mL
  - N=9
Administration of insulin in DKA

Rationale:
- Insulin has NO effect when pH < 6.9 - 7.0
- Cardiac function rapidly worsens with more severe acidosis
- Too rapid correction of acidosis causes metabolic dysbalance and increases acidosis in the brain (bicarbonate cannot cross the BBB)
- $\text{HCO}_3^-$ stimulates $\text{CO}_2$ formation and intracellular acidosis, retards intracellular recovery

Consensus:
- Give $\text{HCO}_3^-$ when pH < 7.0 (50-100 mmol $\text{HCO}_3^-$)
- Just give so much that pH just returns above 7.0
Diabetic ketoacidosis (DKA) – treatment: phosphate

- Phosphate is lost through osmotic diuresis
- With serum $PO_4^- < 0.3$ mmol/l, there is a risk of:
  - muscle weakness
  - haemolysis
  - shift of the $O_2$-dissociation curve
  - reduced cardiac pump function
  - respiratory failure
  - encefalopathy

Consensus:
When $PO_4^- < 0.3$ mmol/l or one of the above: give $KPO_4$ 20-30 mmol/1000ml infusion fluid

Potassium balance in DKA

- Potassium is dominantly intracellular
- Urinary losses occur during evolution of DKA (due to glycosuria)
- Total body potassium stores are greatly reduced in any patient with DKA
- Potassium moves from inside the cell to the extracellular space (plasma)
  - During insulin deficiency
  - In presence of high blood glucose
  - As cells buffer hydrogen ions
- Blood levels of potassium prior to treatment are usually high but may drop precipitously during therapy
Diabetic ketoacidosis (DKA) - treatment: potassium repletion

- Life-threatening hypokalemia can develop during insulin treatment
- Potassium reenters cells with insulinization and correction of acidosis
- The small extracellular compartment experiences a precipitous drop of potassium concentration
- *Anticipatory potassium replacement* during treatment of DKA is almost always required

Diabetic ketoacidosis (DKA) - treatment: potassium repletion

- $K^+ > 5.2$ mEq/L
  - Do not give $K^+$ initially, but check serum $K^+$ with basic metabolic profile every 2 h
  - Establish urine output ~50 mL/hr
- $K^+ < 3.3$ mEq/L
  - Hold insulin and give $K^+$ 20-30 mEq/hr until $K^+ > 3.3$ mEq/L
- $K^+ = 3.3-5.2$ mEq/L
  - Give 20-30 mEq $K^+$ in each L of IV fluid to maintain serum $K^+$ 4-5 mEq/L
Treatment with IV fluids and dextrose

- For severe hypovolaemia, during the first 1-2 hours (in absence of cardiac compromise), give 1-1.5 L 0.9% NaCl
- After initial volume resuscitation, or for more mild dehydration, use intravenous fluid rate of 250-500 mL/hr
- Compute corrected serum Na
  - For every 100 mg/dL BG elevation, add 1.6 mEq/L to Na value
    - Use 0.45% NaCl if corrected Na normal
    - Use 0.9% NaCl if corrected Na <135
- When BG reaches 200 mg/dL (DKA) or 300 mg/dL (HHS), change to 5% dextrose with 0.45% NaCl at 150-250 mL/hr (ie, clamping blood glucose until anion gap has closed in DKA)

Conventional insulin guidelines

- Initiate the correction of hypovolemic shock with fluids, and correct hypokalemia if present, before starting insulin
- When starting insulin, initially infuse 0.1 to 0.14 units/kg/h
- If plasma glucose does not decrease by 50-75 mg/dl (3-4 mmol/l) in the first hour, increase the infusion rate of insulin
- Continue insulin infusion until anion gap closes
- Initiate subcutaneous insulin at least 2 h before interruption of insulin infusion
Insulin protocol for DKA and HHS

- Even with low-dose insulin therapy\textsuperscript{1,2}
  - Hypokalemia and hypoglycaemia may continue to occur
  - Failure to reduce insulin infusion rate as the blood glucose approaches target may lead to hypoglycaemia
- There is a lag between the change in intravenous insulin infusion rate and the resulting effects\textsuperscript{3}


Continuation of physician orders for DKA and HHS

<table>
<thead>
<tr>
<th>Initiation of insulin drip, monitoring of BG, and termination of insulin drip</th>
</tr>
</thead>
<tbody>
<tr>
<td>Initiate IV insulin infusion using selected or default column assignment. Reassignment to a higher column before 4 hours of treatment requires an MD order. If BG fails to fall each hour during hrs 1-4, notify MD</td>
</tr>
<tr>
<td>Adjust column assignment for DKA or HHS based on column change rules, and adjust drip rate based on BG level</td>
</tr>
<tr>
<td>Measure BG every 1 hour (fingerstick or capillary blood sample using point-of-care glucose monitor)</td>
</tr>
<tr>
<td>If BG is within target range x 4hrs, then measure BG q 2 h. If column reassignment occurs, measure q 1 h</td>
</tr>
<tr>
<td>Record BG results, insulin drip rate changes, and column reassignments on the ICU flow sheet</td>
</tr>
<tr>
<td>Obtain order for SQ insulin to be administered q 1-2 h before discontinuing IV insulin</td>
</tr>
</tbody>
</table>

Algorithm for order to treat patient if BG <70 mg/dL

| If BG is <70 mg/dL, administer 25 ml of Glucose 50% by IV |
| Adjust column assignment to next lower column and use pretreatment BG to assign row |
| Recheck BG in 5 minutes. If BG is <70 mg/dL, repeat administration of 25 ml of Glucose 50% by IV |
When to transition from IV insulin infusion to SC insulin

DKA

- BG < 200 mg/dL and 2 of the following
  - $\text{HCO}_3 \geq 15 \text{ mEq/L}$
  - Venous pH > 7.3
  - Anion gap $\leq 12 \text{ mEq/L}$

HHS

- Normal osmolality and regaining of normal mental status
- Allow an overlap of 1-2 h between subcutaneous insulin and discontinuation of intravenous insulin

Fluid and electrolyte management in HHS

- Treatment of HHS requires more free water and greater volume replacement than needed for patients with DKA
- To avoid heart failure, caution is required in the elderly with preexisting heart disease
- Potassium
  - Usually not significantly elevated on admission (unless in renal failure)
  - Replacement required during treatment

## Causes of morbidity and mortality in DKA

- Shock
- Hypokalemia during treatment
- Hypoglycaemia during treatment
- Cerebral edema during treatment
- Hypophosphatemia
- Acute renal failure
- Adult respiratory distress syndrome
- Vascular thrombosis
- Precipitating illness, including MI, stroke, sepsis, pancreatitis, pneumonia

## Characteristics of DKA and HHS - summary

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<thead>
<tr>
<th>Diabetic Ketoacidosis (DKA)</th>
<th>Hyperglycaemic Hyperosmolar State (HHS)</th>
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<tbody>
<tr>
<td>Absolute (or near-absolute) insulin deficiency, resulting in</td>
<td>Severe relative insulin deficiency, resulting in</td>
</tr>
<tr>
<td>• Severe hyperglycaemia</td>
<td>• Profound hyperglycaemia and hyperosmolality (from urinary free water losses)</td>
</tr>
<tr>
<td>• Ketone body production</td>
<td>• No significant ketone production or acidosis</td>
</tr>
<tr>
<td>• Systemic acidosis</td>
<td></td>
</tr>
<tr>
<td>Develops over hours to 1-2 days</td>
<td>Develops over days to weeks</td>
</tr>
<tr>
<td>Most common in type 1 diabetes, but increasingly seen in type 2 diabetes</td>
<td>Typically presents in type 2 or previously unrecognized diabetes</td>
</tr>
<tr>
<td></td>
<td>Higher mortality rate</td>
</tr>
</tbody>
</table>
Hospital discharges for diabetic ketoacidosis (DKA) in the USA

- In 2005, diagnosis of DKA was present on
  - 120,000 discharges
  - 7.4 discharges per 1000 DM patient population
- There was a higher rate of DKA for persons < age 45
  - 55.4 discharges/1000 DM patient population (1987)
  - 31.6 discharges/1000 DM patient population (2005)

Hospital DKA discharges in the USA

Growth in incidence since 1988

140,000 episodes in 2009

Clinical presentation of hyperglycaemic hyperosmolar state

- Compared to DKA, in HHS there is greater severity of:
  - Dehydration
  - Hyperglycaemia
  - Hypernatremia
  - Hyperosmolality
- Because some insulin typically persists in HHS, ketogenesis is absent to minimal and is insufficient to produce significant acidosis
Clinical presentation of hyperglycaemic hyperosmolar state

Patient Profile
- Older
- More comorbidities
- History of type 2 diabetes, which may have been unrecognized

Disease Characteristics
- More insidious development than DKA (weeks vs hours/days)
- Greater osmolality and mental status changes than DKA
- Dehydration presenting with a shock-like state

Electrolyte and fluid deficits in DKA and HHS

<table>
<thead>
<tr>
<th>Parameter</th>
<th>DKA*</th>
<th>HHS*</th>
</tr>
</thead>
<tbody>
<tr>
<td>Water, mL/kg</td>
<td>100 (7 L)</td>
<td>100-200 (10.5 L)</td>
</tr>
<tr>
<td>Sodium, mmol/kg</td>
<td>7-10 (490-700)</td>
<td>5-13 (350-910)</td>
</tr>
<tr>
<td>Potassium, mmol/kg</td>
<td>3-5 (210-300)</td>
<td>5-15 (350-1050)</td>
</tr>
<tr>
<td>Chloride, mmol/kg</td>
<td>3-5 (210-350)</td>
<td>3-7 (210-490)</td>
</tr>
<tr>
<td>Phosphate, mmol/kg</td>
<td>1-1.5 (70-105)</td>
<td>1-2 (70-140)</td>
</tr>
<tr>
<td>Magnesium, mmol/kg</td>
<td>1-2 (70-140)</td>
<td>1-2 (70-140)</td>
</tr>
<tr>
<td>Calcium, mmol/kg</td>
<td>1-2 (70-140)</td>
<td>1-2 (70-140)</td>
</tr>
</tbody>
</table>

* Values (in parentheses) are in mmol unless stated otherwise and refer to the total body deficit for a 70 kg patient.
Initial laboratory evaluation of hyperglycemic emergencies

- Comprehensive metabolic profile
- Serum osmolality
- Serum and urine ketones
- Arterial blood gases
- If needed: lactate
- CBC
- Urinalysis
- ECG
- Blood cultures when suspicion infection

Laboratory diagnostic criteria of DKA and HHS

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Normal range</th>
<th>DKA</th>
<th>HHS</th>
</tr>
</thead>
<tbody>
<tr>
<td>Plasma glucose, mg/dL</td>
<td>76-115</td>
<td>≥250</td>
<td>≥600</td>
</tr>
<tr>
<td>Arterial pH*</td>
<td>7.35-7.45</td>
<td>≤7.30</td>
<td>&gt;7.30</td>
</tr>
<tr>
<td>Serum bicarbonate, mmol/L</td>
<td>22-28</td>
<td>≤15</td>
<td>&gt;15</td>
</tr>
<tr>
<td>Effective serum osmolality, mmol/kg</td>
<td>275-295</td>
<td>≤320</td>
<td>&gt;320</td>
</tr>
<tr>
<td>Anion gap, † mmol/L</td>
<td>&lt;12</td>
<td>&gt;12</td>
<td>Variable</td>
</tr>
<tr>
<td>Serum ketones</td>
<td>Negative</td>
<td>Moderate to high</td>
<td>None or trace</td>
</tr>
<tr>
<td>Urine ketones</td>
<td>Negative</td>
<td>Moderate to high</td>
<td>None or trace</td>
</tr>
</tbody>
</table>

* If venous pH is used, a correction of 0.03 must be made.
† Calculation: Na⁺ - (Cl⁻ + HCO₃⁻).
Finding the cause and preventing recurrence

Possible precipitating causes or factors in DKA

- Nonadherence to insulin regimen or psychiatric issues
- Insulin error or insulin pump malfunction
- Poor “sick-day” management
- Dehydration
- Infection (intra-abdominal, pyelonephritis, flu)
- Myocardial infarction
- Pancreatitis
- Other endocrinopathy (rare)
- Steroid therapy, other drugs or substances
Predischarge Checklist

- Diet information
- Glucose monitor and strips (and associated prescription)
- Medications, insulin, needles (and associated prescription)
- Treatment goals
- Contact phone numbers
- “Medic-Alert” bracelet
- “Survival Skills” training

Education to prevent DKA

- Recognize symptoms and findings that require contact with a healthcare provider
- Prevent ketoacidosis through self-management skills:
  - Glucose testing
  - Appropriate use of urine acetone testing
  - Appropriate maintenance of insulin on sick days
  - Use of supplemental insulin during illness
- Address social factors
Summary

- DKA and HHS are life-threatening emergencies
- Management involves
  - Attention to precipitating cause
  - Fluid and electrolyte management
  - Insulin therapy
  - Patient monitoring
  - Prevention of metabolic complications during recovery
  - Transition to long-term therapy
- Patient education and discharge planning should aim at prevention of recurrence

Resources

Diabetic Ketoacidosis Hyperglycemic Hyperosmolar State
http://inpatient.aace.com/sites/all/files/Strategies-S3-Hyperglycemic-Emergencies.ppt

ISPAD Clinical Practice Consensus Guidelines 2014 Compendium
Diabetic ketoacidosis and hyperglycemic hyperosmolar state
Addendum on Acidosis

Ketone Bodies in DKA

- Unless β-hydroxybutyrate (β-OH B) is specifically ordered, the ketone bodies are estimated by the nitroprusside reaction in the lab, which measures only acetone and acetoacetate (AcAc)
- Acetone is not an acid
Ketone body equilibrium in DKA

\[ \text{AcAc} \leftrightarrow \beta\text{-OH B} \]
\[ \text{NADH + H}^+ \leftrightarrow \text{NAD}^+ \]

- In DKA, the dominant ketoacid is β-hydroxybutyric acid (β-OH B), especially in cases of poor tissue perfusion/lactic acidosis
- During recovery, the balance shifts to acetoacetic acid (AcAc)

Significance of ketone measurements

- β-hydroxybutyrate can only be measured using specialized equipment not available in most in-house laboratories
- During recovery, results from the nitroprusside test might wrongly indicate that the ketone concentration is not improving or is even getting worse
- The best biochemical indicator of resolution of keto-acid excess is simply the anion gap
- There is no rationale for follow-up ketone measurements after the initial measurement has returned high
Molar ratio of β-OH B to AcAc

<table>
<thead>
<tr>
<th>Condition</th>
<th>Ratio</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal health</td>
<td>2 to 1</td>
</tr>
<tr>
<td>DKA</td>
<td>3-4 to 1</td>
</tr>
<tr>
<td>DKA with high redox state</td>
<td>7.7-7.8 to 1</td>
</tr>
</tbody>
</table>

• Significance: Increase of measured ketones may be misleadingly small in DKA with coexisting lactic acidosis and/or alcoholism


Anion gap metabolic acidosis

• The normal anion gap in mEq/L is calculated as:
  \[ [\text{Na}] - ([\text{Cl}] + [\text{HCO}_3^-]) \]
  \[ [140, 105, 25] \]
• The normal gap is <12 mEq/L
• Causes of anion gap acidosis (unmeasured anions) include:
  – Ketoacidosis (diabetic, alcoholic)
  – Lactic acidosis (lactate [underperfusion, sepsis])
  – Uraemia (phosphates, sulphates)
  – Poisonings/overdoses (methanol, ethanol, ethylene glycol, aspirin, paraldehyde)
• In ketoacidosis, the increase of the anion gap above 12 mEq/L is composed of anions derived from keto-acids
Hyperchloremic metabolic acidosis (Non-anion Gap)

- Hyperchloremic acidosis (ie, expansion acidosis) is common during recovery from DKA due to
  - Fluid replacement with saline (NaCl)
  - Renal loss of HCO₃⁻
- Following successful treatment of DKA, a non-anion–gap acidosis may persist after the ketoacidosis has cleared (ie, after closing of the anion gap)
- Closing of the anion gap is a better sign of recovery from DKA than is correction of metabolic acidosis

Formulas for Estimating Serum Osmolality and Effective Osmolality

<table>
<thead>
<tr>
<th>Osmolality</th>
<th>Effective Osmolality</th>
</tr>
</thead>
<tbody>
<tr>
<td>2 x [Na⁺ mEq/L]</td>
<td>2 x [Na⁺ mEq/L]</td>
</tr>
<tr>
<td>+ [glucose mg/dL] / 18</td>
<td>+ [glucose mg/dL] / 18</td>
</tr>
<tr>
<td>+ [BUN mg/dL] / 2.8</td>
<td></td>
</tr>
<tr>
<td>= Sosm (mosm/Kg H₂O)</td>
<td>= Sosm (mosm/Kg H₂O)</td>
</tr>
</tbody>
</table>