Non-Anion Gap
Metabolic Acidosis

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http://PBFuids.com
@kidney_boy
Which type of RTA causes the most severe acidosis

Your Answer

- Proximal RTA (type 2)
- Distal RTA (type 1)
- Hyperkalemic RTA (type 4)
32 y.o. female with fatigue, weakness and muscle aches

Answer polls:
- Uhura: What is the primary acid base...
- Uhura: What is the appropriate pCO2…

Blood Test Results:
- pH: 7.34
- pCO2: 33
- PO2: 87
- HCO3: 18
32 y.o. female with fatigue, weakness and muscle aches
Determine the primary disorder

7.34 / 87 / 33 / 16

Metabolic Acidosis
Predicting $pCO_2$ in metabolic acidosis: Winter’s Formula

- In metabolic acidosis the expected $pCO_2$ can be estimated from the $HCO_3$

  $$\text{Expected } pCO_2 = (1.5 \times HCO_3) + 8 \pm 2$$

- If the $pCO_2$ is higher than predicted then there is an addition respiratory acidosis
- If the $pCO_2$ is lower than predicted there is an additional respiratory alkalosis
Is the compensation appropriate?

Appropriately compensated metabolic acidosis
What anion is associated with the additional acid?
• Metabolic acidosis is further evaluated by determining the anion associated with the increased $\text{H}^+$ cation.

• In medicine we categorize the anions into:

  - chloride
  - not chloride

Non-Anion Gap Metabolic Acidosis

Anion Gap Metabolic Acidosis

…and differentiate the two based on the anion gap.
Anion gap

\[\text{anions} = \text{cations}\]
Anion gap

\[
\text{Cl}^- \quad \text{Na}^+ \\
\text{HCO}_3^- \quad \text{K}^+ \\
\text{other anions} \quad \text{cations}
\]
Calculating the anion gap

- Anion gap = Na – (HCO₃ + Cl)
- Normal is 12
  - Varies from lab to lab
  - Average anion gap in healthy controls is 6 ±3
- Improving chloride assays have resulted in increased chloride levels and a decreased normal anion gap.
Anion gap = Na\(^{-}\) – (HCO\(_3\)\(^{-}\) + Cl\(^{-}\))

Normal is 12

Varies from lab to lab

Average anion gap in healthy controls is 6 ± 3

Improving chloride assays have resulted in increased chloride levels and a decreased normal anion gap.

Calculating the anion gap

\[
\text{Anion gap} = 139 - (115 + 16)
\]

\[
\text{Anion gap} = 7
\]

Varies from lab to lab

Average anion gap in healthy controls is 6 ± 3

Improving chloride assays have resulted in increased chloride levels and a decreased normal anion gap.

Appropriately compensated non-anion gap metabolic acidosis
Non-anion gap metabolic acidosis
NAGMA

Chloride intoxication
Dilutional acidosis
HCl intoxication
Chloride gas intoxication
Early renal failure

GI loss of HCO$_3^-$
Diarrhea
Surgical drains
Fistulas
Ureterosigmoidostomy
Obstructed ureteroileostomy
Cholestyramine

Renal loss of HCO$_3^-$
Renal tubular acidosis
Proximal
Distal
Hypoaldosteronism
pH = 5.5
Cl = 154 mmol/L

Plasma volume
3 liters
Plasma Cl = 105
Degradation products of PVC packaging

Saline solutions for infusion are frequently supplied in polyvinyl chloride (PVC) packaging. PVC can liberate diethylhexyl phthalate (DEHP), a comprehensively studied and controversial ester that serves as a plasticizing agent. In addition, formic and acetic acid - decomposition products formed by oxidation during autoclaving - have been identified in PVC packaged fluids whilst sterilisation with gamma irradiation generates free radicals that may lead to the formation of hydrochloric acid. Studies comparing (i) the theoretical pH of saline derived using the principles outlined above, (ii) the pH of saline prepared in the laboratory in the absence of PVC, and (iii) clinically used commercial source material revealed that whilst very similar pH to that predicted median pH of the PVC interestingly, the pH of s-
Decreases in bicarbonate force the reaction to the left, replacing the bicarbonate and increasing H⁺

Increases in exogenous acid drive the reaction to the right, bicarbonate is consumed
in both, the end result is an increase in $H^+$ and a decrease in $HCO_3^-$, the defining characteristic of metabolic acidosis.
In NAGMA we will use the “loss of HCO$_3^-$” model
NAGMA

Chloride intoxication
Dilutional acidosis
HCl intoxication
Chloride gas intoxication
Early renal failure

GI loss of $\text{HCO}_3$
Diarrhea
Surgical drains
Fistulas
Ureterosigmoidostomy
Obstructed ureteroileostomy
Cholestyramine

Renal loss of $\text{HCO}_3$
Renal tubular acidosis
Proximal
Distal
Hypoaldosteronism
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<th>Value 2</th>
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<td>90</td>
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<td>Pancreas</td>
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<td>Large intestines</td>
<td>5.0</td>
<td>90</td>
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<tr>
<td></td>
<td>5.0</td>
<td>90</td>
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</table>
Ureterosigmoidostomy

Ureteroileostomy

Urine pH=5.5

Serum pH=7.4

100 fold difference
Urinary diversion

• Urine $\text{NH}_4^+$ is absorbed by the intestinal mucosa and converted to $\text{H}^+$ and $\text{NH}_3$.

• Urine Chloride stimulates colonic expression and activation of $\text{Cl}^-/\text{HCO}_3^-$ exchangers exacerbating the loss of bicarbonate.

• Increased colonic pressures can cause urinary obstruction resulting in a urinary acidification defect
  – Repeated episodes of pyelonephritis may do the same
NAGMA

Chloride intoxication
Dilutional acidosis
HCl intoxication
Chloride gas intoxication
Early renal failure

GI loss of $\text{HCO}_3^-$
- Diarrhea
- Surgical drains
- Fistulas
- Ureterosigmoidostomy
- Obstructed ureteroileostomy
- Cholestyramine

Renal loss of $\text{HCO}_3^-$
- Renal tubular acidosis
  - Proximal
  - Distal
- Hypoaldosteronism
Renal causes of NAGMA:
Renal Tubular Acidosis (RTA)

- RTA can be due to a failure of the kidney to

  1. *Reabsorb* all of the filtered bicarbonate

  2. *Synthesize* new bicarbonate to replace bicarbonate lost to metabolism

  3. *Stow* hydrogen ions in ammonia so we can clear the daily acid load
The kidney’s role in $\text{HCO}_3^-$ handling is not excretory.
The kidney’s role in HCO$_3$ handling is not excretory, rather the kidney needs to reabsorb the thousands of millimoles of filtered bicarbonate.
The kidney’s role in HCO$_3$ handling is not excretory, rather the kidney needs to reabsorb the thousands of millimoles of filtered bicarbonate and synthesize a few dozen millimoles of bicarbonate to replace the bicarbonate lost to the daily acid load.
Reabsorption
How much bicarbonate is filtered every day?

- Normal plasma HCO$_3$ concentration is 24 mmol/L
- Normal GFR is 100 mL/min, or 0.1 L per minute
- 1440 minutes in a day
- $24 \times 0.1 \times 1440 = 3,456$ mmol of bicarbonate are filtered a day (filtered load)
Synthesis
How much bicarbonate needs to be replaced?
How much bicarbonate is consumed?
*The daily acid load*

- Protein metabolism consumes bicarbonate
  - 1 mmol/kg
  - 2 mmol/kg in children
  - 4 mmol/kg in infants

- This bicarbonate must be replaced to maintain homeostasis
Two jobs, two locations

- The proximal tubule needs to reabsorb 3,456 mmol of bicarbonate that is filtered every day.
- Proximal tubule

3456 mmol/day

- The kidney must synthesize 50-100 mmol per day of new HCO$_3^-$ to replace HCO$_3^-$ lost buffering the daily acid load.
- Cortical collecting tubule

50-100 mmol/day
Overview: The proximal tubule normally functions to resorb water and solutes.

Sodium flows down its concentration gradient into the cell.

Resorption of sodium provides the energy to resorb filtered solutes.

The resorption of sodium provides the energy for hydrogen secretion.

Intracellular sodium concentration is kept low by the Na-K-ATPase pump.
Proximal tubule: reabsorption of filtered bicarbonate
Bicarbonate handling

- The proximal tubule needs to reabsorb 3,456 mmol of bicarbonate that is filtered every day.
- Proximal tubule

- The kidney must synthesize 50-100 mmol per day of new HCO$_3^-$ to replace HCO$_3^-$ lost buffering the daily acid load.
- Cortical collecting tubule

3456 mmol/day

50-100 mmol/day
Distal tubule, completion of reabsorption and replacing bicarbonate lost to the daily acid load.

3 step process:

Electrogenic movement of sodium into the tubular cells (eNaC)

H$^+$ pumped into the tubular lumen ATPase

Maintain the 1000 fold concentration gradient
Fate of excreted hydrogen ion

The minimal urine pH is 4.5. This is a $\text{H}^+$ concentration a 1000 times that of plasma.

But it still is only 0.04 mmol/L.

In order to excrete 50 mmol (to produce enough bicarbonate to account for the daily acid load) one would need...

1,250 liters of urine.
Fate of excreted hydrogen ion

- $\text{NH}_4^+$ (Ammonium)
- $\text{H}_2\text{PO}_4^-$ (Titratable acid)

Diagram:
- ATP
- AMP
- $\text{H}^+$
• Excretion of the daily acid load as free hydrogen is limited by a minimum urinary pH of 4.5
  – Only 0.1% of the daily acid load is excreted this way

• Titratable acid is urinary phosphate
  – Titratable acid carries a significant portion of the daily acid load
  – Limited by dietary phosphate
  – Does not respond to changes in the acid load

• Ammonium carries the bulk of the daily acid load
  – In response to an acid load the kidney will increase production of ammonia (NH₃) in order to accept additional protons to carry the load
3 steps in renal bicarbonate handling

3456 mmol/day

50-100 mmol/day

\[ \text{NH}_4^+ \rightarrow \text{NH}_3 + \]n

\[ \text{H}_2\text{PO}_4^- \rightarrow \text{HPO}_4^{2-} + \]n
Each step can fail which causes RTA and NAGMA.
Proximal RTA (Type 2)

- The Tm is the maximum plasma concentration of any solute at which the proximal tubule is able to completely reabsorb the solute.

- Beyond the Tm the substance will be incompletely reabsorbed and spill in the urine.

- In Proximal RTA the Tm for bicarbonate is reduced from 26 to 15-20 mmol/L.
Proximal RTA (Type 2)

Damage to the proximal tubule decreases its Tm from 28 to somewhere in the mid-teens

Tm for HCO₃ at 15

Serum HCO₃ is > Tm so HCO₃ spills into the urine
Proximal RTA (Type 2)

Serum $\text{HCO}_3^-$ then falls
When it falls to the Tm (15 mmol/L) the kidney appears to work normally
Homeostasis resumes but at a decreased $\text{HCO}_3^-$
Proximal RTA (Type 2)

If the patient encounters an acid load, they synthesize new bicarbonate to return the serum HCO$_3^-$ to altered Tm (15)
Proximal RTA (Type 2)

During treatment with exogenous bicarbonate the serum bicarbonate will rise above the Tm and the patient will spill bicarbonate
Proximal RTA: etiologies

- **Acquired**
  - Acetylozolamide
  - Ifosfamide
  - Chronic hypocalcemia
  - Multiple myeloma
  - Cisplatin
  - Lead toxicity
  - Mercury poisoning
  - Streptozocin
  - Expired tetracycline

- **Genetic**
  - Cystinosis
  - Galactosemia
  - Hereditary fructose intolerance
  - Wilson’s disease

- **Hyperparathyroidism**
  - Variable

- **Chronic respiratory alkalosis**
  - Intracellular alkalosis
Respiratory alkalosis

- The pCO$_2$ falls due to the primary pathology
- The bicarbonate falls to compensate
  - The Tm for bicarbonate is lowered
Proximal RTA: consequences

- Loss of potassium (hypokalemia)
- Bone disease
  - Bone buffering of the acidosis
- Decreased growth
- Not typically complicated by stones
Each step can fail which causes RTA and NAGMA.

Proximal RTA

3456 mmol/day

Distal RTA

50-100 mmol/day

Hyperkalemic RTA
Distal RTA, the Murphy’s Law of $\text{HCO}_3^-$ handling
Distal RTA, the Murphy’s Law of distal HCO₃ handling
Distal RTA (Type 1)

Failure can happen at any one of the three steps in urinary acidification
Distal RTA: Voltage dependent

- Only variety of distal RTA which is hyperkalemic
- Differentiate from type 4 by failure to respond to fludrocortisone.
  - Obstructive uropathy
  - Sickle cell anemia
  - Lupus
  - Triameterene
  - Amiloride
Distal RTA: H⁺ Secretion

- Called classic distal RTA
- Most common cause of distal RTA
  - Congenital
  - Lithium
  - Multiple myeloma
  - Lupus
  - Pyelonephritis
  - Sickle cell anemia
  - Sjögren’s syndrome
  - Toluene (Glue sniffing)
  - Wilson’s disease
Distal RTA: Gradient defect

- Amphotericin B
Distal RTA: consequences

- **Bones**
  - Chronic metabolic acidosis results in bone buffering.
    - Bicarbonate
    - Phosphate
    - Calcium
  
- **Kidney stones**
  - Calcium phosphate stones
    - Due to hypercalciuria
    - Increased urine pH
    - Decreased urinary citrate

Well Mr. Osborne, it may not be kidney stones after all.
Each step can fail which causes RTA and NAGMA.
Chronic hyperkalemia of any etiology decreases ammoniagenesis

- $\text{NH}_3$ is needed to excrete excess $\text{H}^+$ in the urine
Intracellular acidosis stimulates intra-renal ammonia production
Intracellular alkalosis suppresses intra-renal ammonia production

With increases in serum potassium, potassium shifts inside the cells

To maintain electroneutrality, H^+ moves out of the cells
Hypoaldosteronism: Type 4

- Chronic hyperkalemia decreases ammoniagenesis
- Without ammonia acid excretion is modest
- Urinary acidification is intact
- Acidosis is typically mild without significant bone or stone disease
- Primary problem is high potassium
Diagnosing the cause of:

non-anion gap metabolic acidosis
To look for renal H\(^+\) clearance look for urinary ammonium

\[
\text{NH}_4^+ \leftarrow \text{NH}_3 + \text{H}^+
\]

Ammonium

\[
\text{H}_2\text{PO}_4^- \leftarrow \text{HPO}_4^{2-} + \text{H}^+
\]

Titratable acid

\[
\text{ATP} \rightarrow \text{AMP} \rightarrow \text{H}^+
\]
Urinary anion gap: \((\text{Na}^+ + \text{K}^+) - \text{Cl}^-\)

- In the presence of ammonium the chloride will be larger than the sum of Na and K.
- So a negative anion gap means ammonium in the urine.
- **Ammonium in the urine = effective renal acid secretion**
- Ammonium in the urine usually rules out RTA
Urinary anion gap:  \((\text{Na}^+ + \text{K}^+) - \text{Cl}^-\)

Urinary ammonium detector

- In the absence of ammonium the Na and K will be larger than the chloride.
- So a positive anion gap means no ammonium in the urine.
- **No ammonium in the urine = no effective renal acid secretion, regardless of the urinary pH**
- A positive urinary anion gap in the presence of metabolic acidosis is consistent with an RTA
Answer polls:

- Urinary anion gap and diarrhea
- Urinary anion gap and proximal RTA
- Urinary anion gap and distal RTA
- Urinary anion gap and RTA type 4
NAGMA and urinary anion gap

- **Diarrhea**
  - Negative
- **Proximal RTA**
  - At baseline
    - Negative (variable)
  - During treatment
    - Positive
  - During acid load
    - Negative
- **Distal RTA:**
  - Positive
- **Type IV RTA**
  - Positive
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<th>Age</th>
<th>Gender</th>
<th>Presentation</th>
<th>Blood Pressure</th>
<th>Heart Rate</th>
<th>Temperature</th>
<th>WBC</th>
<th>RBC</th>
<th>Hemoglobin</th>
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<td>32 y.o. female</td>
<td>with fatigue, weakness and muscle aches</td>
<td>139/115</td>
<td>3.1</td>
<td>17</td>
<td>1.0</td>
<td>7.34</td>
<td>87</td>
<td>33</td>
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</table>
 Appropriately compensated non-anion gap metabolic acidosis due to distal RTA
74 y.o. female with 34 year history of DM c/o weakness

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Answer polls:

• Dowager Countess: What is the primary acid-base...
• Dowager Countess: What is the appropriate pCO2...
• Dowager Countess: What additional acid-base disorders are present

139 123 21

6.6 17 1.2

7.34 / 87 / 33 / 16

Albumin 1.8
74 y.o. female with 34 year history of DM c/o weakness

<table>
<thead>
<tr>
<th>139</th>
<th>123</th>
<th>21</th>
<th>7.34 / 38 / 92 / 16</th>
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<tbody>
<tr>
<td>6.6</td>
<td>17</td>
<td>1.2</td>
<td>Albumin 1.8</td>
</tr>
</tbody>
</table>
Determine the primary disorder

1. Acidosis or alkalosis
   - If the pH is less than 7.4 it is acidosis
   - If the pH is greater than 7.4 it is alkalosis

2. Determine if it is respiratory or metabolic
   - If the pH, bicarbonate and pCO\_2 all move in the same direction (up or down) it is metabolic
   - If the pH, bicarbonate and pCO\_2 move in discordant directions (up and down) it is respiratory

Metabolic Acidosis
Predicting pCO$_2$ in metabolic acidosis: Winter’s Formula

\[
\text{Expected } pCO_2 = (1.5 \times HCO_3^-) + 8 \pm 2
\]

Actual pCO$_2$ is 38, which is higher than predicted, so the patient has an additional respiratory acidosis.

Combined metabolic acidosis and respiratory acidosis
Calculating the anion gap

\[
\begin{align*}
\text{Na} - (\text{HCO}_3^- + \text{Cl}) &= 139 - (123 + 17) \\
&= 139 - 140 \\
&= -1
\end{align*}
\]

A negative anion gap! That’s got to mean something!
Hypoalbuminuria, hypophosphatemia

- The “other anions” includes phosphate and albumin
- Hypoalbuminuria and hypophosphatemia lowers the anion gap
- If one fails to adjust the upper and lower limit of the normal anion gap, altered albumin and phosphorous can hide a pathologic anion gap

- To estimate the normal anion gap for any individual multiply the albumin by 2.5 and add half the phosphorous
Other causes of a low anion gap

- Increased chloride
  - Hypertriglyceridemia
  - Bromide
  - Iodide
- Decreased “Unmeasured anions”
  - Albumin
  - Phosphorous
  - IgA
- Increased “Unmeasured cations”
  - Hyperkalemia
  - Hypercalcemia
  - Hypermagnesemia
  - Lithium
  - Increased cationic paraproteins
    - IgG

\[ \text{Sodium} \quad \text{Chloride} \quad \text{Bicarb} \]
\[ \text{Potassium} \quad \text{Calcium} \quad \text{Magnesium} \quad \text{IgA} \]

\[ \text{Normal Anion gap} \]
Recent lab history

Albumin falls 2 g/dL
Potassium climbs 2 mmol/L
Anion Gap falls 7 from 6 to -1
Diagnose the cause of:

**non-anion gap metabolic acidosis with hyperkalemia**

1. **Type four RTA, hyporenin-hypoaldo**
2. **Hyperkalemic Distal (Type 1) RTA, voltage dependent distal RTA**
A dipstick for aldosterone activity: The trans-tubular potassium gradient The TTKG
The TTKG is not useful in the diagnosis of hyperkalemia.

- Persistent hyperkalemia is always associated with an inappropriately low TTKG.

The TTKG can be helpful in differentiating renal from extra-renal potassium losses in hypokalemia.

Recent data on solute handling in the medullary collecting duct calls into question the physiologic assumptions required for the TTKG to be valid.

- [PubMed link](http://www.ncbi.nlm.nih.gov/m/pubmed/21788894/)
Trans-tubular Potassium Gradient (TTKG)

- The ratio of tubular to venous K indicates the level of aldosterone activity.
- In the presence of hyperkalemia the ratio should be > 10.
- In the presence of hypokalemia the ratio of < 4.
Trans-tubular Potassium Gradient (TTKG)

• The trans-tubular potassium gradient adjusts the urine potassium for water loss in the collecting ducts.

• This allows the use of urinary potassium to calculate the ratio of potassium from the tubule to the interstitium in the CCD.

\[
TTKG = \frac{K_{\text{CCD}}}{K_{\text{plasma}}}
\]
Trans-tubular Potassium Gradient (TTKG)

- Pre-requisites to using the TTKG as a measure of aldosterone activity:
  - Urine osmolality > serum osmolality
  - Urine Na > 20 mmol/L
1. **Type four RTA, hyporenin-hypoald**
   - Low TTKG
   - Low aldosterone

2. **Hyperkalemic Distal (Type 1) RTA, voltage dependent distal RTA**
   - Low TTKG
   - High aldosterone
74 y.o. female with 34 year history of DM c/o weakness

Both the bicarbonate and potassium were normal at admission. This is hospital acquired RTA (type 1 or 4)
1. **Type four RTA, hyporenin-hypoaldo**
   - Spironolactone
   - ACEi/ARB
   - Heparin

2. **Hyperkalemic Distal (Type 1), voltage dependent distal RTA**
   - Amiloride
   - Triamterene
   - Trimethoprim
1. **Type four RTA, hyporenin-hypoaldosterone**
   - Spironolactone
   - ACEi/ARB
   - Heparin

2. **Hyperkalemic Distal (Type 1), voltage dependent distal RTA**
   - Amiloride
   - Triamterene
   - Trimethoprim

**Let's play: Name that drug!**
74 y.o. female with 34 year history of DM and c/o weakness

Patient’s TTKG was 2.7 with a K of 5.7

Aldosterone was 22

The patient had been started on a high dose of TMP/SMX for a partially resistant urinary tract infection

2. Hyperkalemic Distal (Type 1), voltage dependent distal RTA
Two women with non-anion gap metabolic acidosis
One with hypokalemia
One with hyperkalemia
Both with distal RTA
Lets do some cases

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Lets do some cases
25 year old man
CC: Recurrent kidney stones

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<th>serum</th>
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<td>112</td>
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<tr>
<td>3.5</td>
<td>17</td>
<td>1.2</td>
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</table>

urine pH 6.5

Type 1 classical distal RTA

Answer poll: 25 year old recurrent kidney stones
36 yr old. Diabetes since age 12, retinopathy, neuropathy. On insulin and ACEi

\[
\begin{array}{c|c|c}
142 & 112 & 32 \\
6.0 & 20 & 1.9 \\
\end{array}
\]

urine pH 6.5

Answer poll: 36 year old diabetic
25 year old man
CC: Recurrent kidney stones

serum

\[
\begin{array}{c|c|c}
137 & 112 & 14 \\
3.5 & 17 & 1.2 \\
\end{array}
\]

urine

\[
\begin{array}{c|c}
55 & 73 \\
23 & \\
\end{array}
\]

urine pH 6.5

Type 1 classical distal RTA
36 yr old. Diabetes since age 12, retinopathy, neuropathy. On insulin and ACEi

\[
\begin{array}{ccc}
142 & 112 & 32 \\
6.0 & 20 & 1.9 \\
\end{array}
\]

urine pH 5.5

Type 4 RTA

What do you do?
36 yr old. Diabetes since age 12, retinopathy, neuropathy. On insulin but not an ACEi

\[
\begin{array}{c|c|c|c|c}
142 & 112 & 32 \\
4.7 & 23 & 1.9 \\
\end{array}
\]

urine pH 5.5

Type 4 RTA
36 yr old. Diabetes since age 12, retinopathy, neuropathy. On insulin and ACEi

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urine pH 6.5

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7.31 / 30 / 115 / 15

Answer poll: same 36 year old, 6 months later
36 yr old. Diabetes since age 12, retinopathy, neuropathy. On insulin but not an ACEi

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urine pH 5.5

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<tr>
<td>6.2</td>
<td>10</td>
<td>600</td>
<td>2.6</td>
<td></td>
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<tr>
<td>135</td>
<td>115</td>
<td>36</td>
<td></td>
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<tr>
<td>4.8</td>
<td>14</td>
<td>300</td>
<td>2.0</td>
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</table>

7.31 / 30 / 115 / 15
Type 4 RTA

Top left is type 4 RTA

Bottom left is same patient with addition of diuretic

Top right is AGMA due to DKA

Bottom right is NAGMA in the recovery phase of DKA with dilutional acidosis
45 year old with alcohol abuse. Also c/o occasional diarrhea.

\[
\begin{array}{c|c|c|c|c}
132 & 107 & 10 \\
3.2 & 17 & 0.6 \\
\end{array}
\]

Albumin 2.9

7.44 / 28 / 96 / 19

Answer poll: 45 year old alcoholic
45 year old with alcohol abuse. Also c/o occasional diarrhea.

\[
\begin{array}{c|c|c}
132 & 107 & 10 \\
3.2 & 17 & 0.6 \\
\end{array}
\]

Albumin 2.9

7.44 / 28 / 96 / 19
22 y.o. AA female with hyperkalemia and microscopic hematuria

<table>
<thead>
<tr>
<th>Blood Pressure</th>
<th>Blood Urea Nitrogen</th>
<th>Creatinine</th>
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<tbody>
<tr>
<td>140/115</td>
<td>6.0</td>
<td>1.2</td>
</tr>
</tbody>
</table>

**Urinalysis**
- pH 6.5
- + Blood
- + WBC
- 20-50 RBC
- 1 RBC cast

Systemic Lupus Erythematosus

**Electrogenic Distal RTA**

Answer poll: 22 y.o. with microscopic hematuria
22 y.o. AA female with hyperkalemia and microscopic hematuria

**Electrogenic Distal RTA**

**Blood Pressure**
- SBP: 140
- DBP: 115
- HR: 18

**Electrolytes**
- Potassium: 6.0
- Sodium: 18
- Chloride: 1.2

**Urinalysis**
- Specific gravity: 1.015
- pH: 6.5
- Blood
- WBC: +
- RBC: 20-50
- 1 RBC cast

**Systemic Lupus Erythematosus**
Most common error in acid-base

Personal observation
AE

- 66 yo white male
- PMHx DM, paraplegia 2° MVA
- Klebsiella urosepsis induced ARF
- Blood Cxrs + for Klebsiella
<table>
<thead>
<tr>
<th>Date</th>
<th>pH</th>
<th>pCO₂</th>
<th>BE</th>
<th>K+</th>
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</thead>
<tbody>
<tr>
<td>8/16</td>
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<td>58</td>
<td>31</td>
<td>25</td>
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<td>5.4</td>
<td>20</td>
<td>1.2</td>
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<td>21</td>
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<td>16</td>
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<td>Start bicarbonate gtt</td>
</tr>
</tbody>
</table>

- Start oral bicarbonate
Done. Thank-you

Please do the post test and answer the discussion question