Renal Tubular Acidosis: A Drug-Centric Perspective

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- **leflunomide, ilotiapam, osalplatin, oteferoxir acetazolamid amiloridaprid cyclopamine, tacrolimus adefovir/tenofovir/cidofovir/disulfiram/ lamivudine / stavudine 5-AZA coamnirin streptozocin letictamides sulfonate**

### Alkaline Replacement:

<table>
<thead>
<tr>
<th>Non-drug causes:</th>
<th>Primary, hereditary, sporadic, decreased carbonic anhydrase activity/CA deficiency, genetic diseases</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Fancconi syndrome</strong>, tyrosinemia, Wilson's disease, glycogen storage disease, galactosemia, ARC Syndrome, Alport syndrome, multiple myeloma, amyloidosis, nephropthic syndrome, hypocalcemia, vit.D deficiency / resistance, Sjogren's syndrome, renal vein thrombosis, renal transplant rejection, lead / cadmium / mercury toxicity, hyperparathyroidism, medullary cystic kidney disease</td>
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</tr>
</tbody>
</table>

- **Furosemide 50-200 mcg/d**
- **K+ restriction**
- **K+ supps may be needed**

### Goals of therapy:

1. prevent nephrocalcinosis, osteomalacia, rickets
2. prevent severe acidosis, respiratory failure & death
3. prevent hyperkalemic paralysis with (rhabdomyolysis), hyperkalemia & death
4. prevent irreversible renal damage (if drug-induced)
5. prevent fatigue

### NOTES:

- UAG = urine (Na + K) - urine Cl
- urine pH must be measured promptly (it increases as it sits since CO2 evaporates & bacteria produce urea) - increased urine pH can’t run pH on previously obtained sample
- most labs measure pH in 0.5 increments and only if >5.5
- urine Cl is hard to do (not all labs can do it)

### Alkali Replacement:

- Total body bicarb deficit (mEq)= (desired - actual) x 0.6 x wt
- Baking soda contains 12 mEq bicarb/kg and ~270 mg Na+/g
- K-citrate & Na-citrate (Dicitrate) contain 1mEq bicarb/mL (+ 1mEq K+ or Na+ per mL)
- NaBicarb tablets contain 325mg NaBicarb (=4 mEq bicarb + 90 mg Na+/tablet)

### Type 2 RTA (proximal RTA, pRTA):

- decreased bicarb resorption in proximal tubule (threshold >15)
- Don’t forget about NH3 synthesis/transfer defect!
- Features: urine pH<5.5, normokalemia

### Type 1 RTA (distal RTA, dRTA):

- permeability defect
- ? back-diffusion of H+ out of tubule
- Features: urine pH<5.5, hypokalemia

### Type 4 RTA!

- Hyperkalemia causes 1 NH3 synthesis → 1 ability to acidify urine...
- Features: hypokalemia urine ph<5.5 when acidic

<table>
<thead>
<tr>
<th>Renal Tubular Acidosis Comparison Table</th>
</tr>
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<tbody>
<tr>
<td>Type</td>
</tr>
<tr>
<td>------</td>
</tr>
<tr>
<td>pRTA (Type 2)</td>
</tr>
<tr>
<td>dRTA (Type 1)</td>
</tr>
<tr>
<td>-NH3 synth / transport defect</td>
</tr>
<tr>
<td>-secretory defect</td>
</tr>
<tr>
<td>-permeability defect</td>
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<tr>
<td>-voltage defect</td>
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<tr>
<td>Type 4</td>
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</tbody>
</table>

†currently thought to be due to overactive Cl-/HCO3-exchange pump in distal tubule (in Type1) [Pediatr Nephrol 2006;21:206-211] *when corrected for acidosis (eg, pH Δ 0.1 + K+ Δ 0.6 mmol/L) **UAG = urine (Na + K) – Cl

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[Image 203x244 to 260x257]