



# TYPE IV RENAL TUBULAR ACIDOSIS

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# OVERVIEW: TYPE IV RTA

- ❖ Most common form of RTA
- ❖ Normal anion gap metabolic acidosis with hyperkalemia
- ❖ Urine pH < 5.5, mild renal insufficiency
- ❖ Impaired ammonia synthesis → ↓  $\text{NH}_4^+$  excretion → Positive UAG
- ❖ Tubular disorder (Chronic tubulointerstitial disease)
- ❖ New term: *Hyperkalemic RTA / Tubular Hyperkalemia*

# ETIOLOGY

## ❖ Chronic Tubulointerstitial Causes

- Analgesic nephropathy
- Obstructive nephropathy
- Sickle cell nephropathy
- Lead nephropathy
- Diabetes mellitus

❖ ~50% of Type IV RTA occurs in diabetic patients

## ❖ Other Associations

- Chronic kidney disease
- Primary adrenal insufficiency

## ❖ Genetic causes:

- Congenital hypoaldosteronism (21-hydroxylase deficiency)
- Pseudohypoaldosteronism type 2 (Gordon's syndrome)

# PATHOPHYSIOLOGY

- ❖ **Hyporeninemic hypoaldosteronism** (commonest mechanism)
- ❖  $\downarrow$  **Renin and/or aldosterone**  $\rightarrow$   $\downarrow$  **distal  $\text{Na}^+$  reabsorption**  $\rightarrow$   $\downarrow$   **$\text{K}^+$  and  $\text{H}^+$  secretion**
- ❖  $\downarrow$  **Cortical collecting tubule responsiveness**
- ❖ **Impaired  $\text{NH}_4^+$  synthesis**  $\rightarrow$   $\downarrow$  **acid excretion**
- ❖ **Result:** Hyperkalemia + normal anion gap metabolic acidosis

# URINE INDICES

- ❖ **Urine Anion Gap (UAG):**
- ❖  $(U Na^+ + U K^+) - U Cl^-$
- ❖ Normal:  $-50$  to  $-20$  mEq/L
- ❖ Indirect measure of **urinary  $NH_4^+$  excretion**
- ❖ **GI  $HCO_3^-$  loss  $\rightarrow \uparrow NH_4^+ \rightarrow$  UAG negative**
- ❖ **RTA  $\rightarrow \downarrow NH_4^+ \rightarrow$  UAG positive**
- ❖ **Transtubular  $K^+$  Gradient (TTKG):**
- ❖ **Low in Type IV RTA ( $\downarrow$  distal  $K^+$  secretion)**





# DRUG-RELATED CAUSES

ACE inhibitors

ARBs

Mineralocorticoid receptor antagonists (spironolactone, eplerenone)

Direct renin inhibitors

$\beta$ -blockers

NSAIDs

Calcineurin inhibitors (cyclosporine, tacrolimus)

Heparin & analogues

Trimethoprim, herbal preparations

# MANAGEMENT

## ❖ General Measures

- ❖ Stop or reduce offending drugs (NSAIDs, ACEi/ARB, MRAs, etc.)
- ❖ Manage hyperkalemia: Calcium gluconate, insulin, newer K<sup>+</sup> binders
- ❖ Treat hyperglycemia
- ❖ Monitor ECG; temporary HD if severe


## ❖ Specific Measures

### ❖ Fludrocortisone 0.1–0.2 mg/day (selected cases)

- Watch for HTN, edema, alkalosis
- Avoid in volume overload or heart failure

### ❖ Before RAASi initiation:

- Check eGFR, S. Cr, S. K<sup>+</sup>
- Review every 2 weeks initially



*Type IV RTA is common in diabetics,  
potentiated by RAAS blockade, and diagnosed  
via UAG/TTKG — respond well to drug  
withdrawal,  $K^+$  correction, and cautious  
fludrocortisone use.*





THANK YOU