

Hyperchloremic metabolic acidosis: causes

Definition: A normal AG acidosis is characterized by a lowered bicarbonate concentration, which is counterbalanced by an equivalent increase in plasma chloride concentration, **AKA hyperchloremic metabolic acidosis.**

The term hyperchloremic acidosis (ie, RTA) refers to a diverse group of tubular disorders, uncoupled from glomerular damage, characterized by impairment of urinary acidification without urea and anion retention. Consequently, typically RTA is unaccompanied by significant decreases in GFR. These disorders can be divided into 2 general categories, proximal (type II) and distal (types I and IV).

Mechanism	↑AG	↓AG
↑acid production	Lactic acidosis Ketoacidosis (DM, starvation, ETOH) Ingestions (Methanol, Ethylene glycol, ASA, Toluene if early or kidney impaired, Diethylene glycol, Propylene glycol) D-lactic acidosis Pyroglutamic acid	Toluene if late and renal fx preserved
Loss of HCO ₃		Diarrhea or GI loss by tube drainage Type 2 RTA Post Rx of ketoacidosis Carbonic anhydrase inhibitors Ureteral diversion (ileal loop)
↓renal acid excretion	CKD	CKD and tubular dysfx (but preserved GFR) Type 1 RTA Type 4 RTA (hypoaldosteronism)

Characteristics of different types of RTA:

	Type 1 RTA	Type 2 RTA	Type 4 RTA
Primary defect	↓ distal acidification	↓HCO ₃ prox reabsorb	↓aldosterone secretion
Plasma HCO ₃ (meq/L)	Variable but gen < 10	12-20	Variable but gen >17
Urine pH	> 5.3	variable	Variable
Plasma K	Usually ↓	↓	↑