



### Ethylene Glycol Toxicity (2)

Component of antifreeze and solvents.

Symptoms: Neurologic and cardiopulmonary abnormalities, flank pain, and renal failure.

Needle shaped oxalate crystals in urine.

Treatment:

-Administration of ethanol or fomepizole

-Administration of folic acid, thiamine, and pyridoxine.

-Hemodialysis for removal of toxic metabolites and parent compound.

### Lactic Acidosis (2)

Cause: Increase in lactate production or Decrease in lactate utilization.

Treatment:

-Correction of the underlying disorder & reversal of circulatory failure is the 1ry therapy.

-NaHCO<sub>3</sub> administration indicated if severe acidosis pH <7.1 or loss of buffering capacity (HCO<sub>3</sub><5 mEq/L).

-Hemodialysis may be indicated in resistant cases.

-Alternative therapies:

Carbicarb: equimolar mixture of sodium carbonate and sodium bicarbonate.

Dichloroacetate: activates pyruvate dehydrogenase and increases oxidation of pyruvate.

### Renal (2)

(UAG +ve)

	Distal (RTA Type 1)	Proximal (RTA Type 2)	Hyporeninemic/ Hypoaldosteronism (RTA Type 4)
Cause	<ul style="list-style-type: none"><li>• Idiopathic</li><li>• Familial</li><li>• Sjogren syndrome</li><li>• Hypercalciuria,</li><li>• RA,</li><li>• SCA,</li><li>• Amphotericin</li></ul>	<ul style="list-style-type: none"><li>• Idiopathic,</li><li>• Multiple myeloma</li><li>• Carbonic anhydrase inhibitor</li><li>• Heavy metals</li><li>• Hypocalcemia,</li><li>• Vit D deficiency</li></ul>	<ul style="list-style-type: none"><li>• Diabetes</li><li>• ACE inhibitor</li><li>• Tubulointerstitial nephritis</li><li>• NSAIDS</li><li>• Heparin</li><li>• Adrenal insufficiency (Obstrucuropathy)</li><li>• K+sparing diuretics</li></ul>
Defect	Impaired distal tubule H <sup>+</sup> excretion	Impaired proximal tubule HCO <sub>3</sub> <sup>-</sup> absorption +/- glycosuria, aminoaciduria, phosphaturia	Aldosterone deficiency/ resistance
Plasma HCO <sub>3</sub> <sup>-</sup>	Variable; usually severe < 10 mEq/L	Less severe, 12-20 mEq/L	> 15 mEq/L
Urine PH	> 5.3	> 5.3 if serum HCO <sub>3</sub> <sup>-</sup> above reabsorptive threshold < 5.3 if serum HCO <sub>3</sub> <sup>-</sup> below reabsorptive threshold	< 5.3

Plasma K <sup>+</sup>	Low	Low	High
UAG	+ve	Variable	+ve
Associated Condition	Renal stones	<ul style="list-style-type: none"> <li>• Rickets</li> <li>• Osteomalacia</li> <li>• Fanconi syndrome</li> </ul>	None
Treatment	Alkali therapy	Alkali therapy or Thiazide (in resistant cases)	Treat the cause

### Note

#### IV HCO<sub>3</sub> (1)

Isotonic sodium bicarbonate 1.26%. (HCO<sub>3</sub><sup>-</sup> =150 & Na<sup>+</sup> = 150)

Hypertonic sodium bicarbonate 8.4%. (HCO<sub>3</sub><sup>-</sup> =1000 & Na<sup>+</sup> = 1000)

-Isotonic (1.26%) sodium bicarbonate may be used to correct acidosis associated with renal failure or to induce a forced alkaline diuresis.

-The hypertonic (8.4%) solution (1mEq HCO<sub>3</sub><sup>-</sup> /mL) is rarely required in intensive care practice to raise blood pH in severe metabolic acidosis.

-Excessive administration may cause hyperosmolality, hypernatraemia, hypokalaemia & sodium overload.

-Fluid and potassium deficit should be corrected first.

- Both arterial and venous values should be monitored. (2)

-Continuous or intermittent hemodialysis may also be used to correct severe, refractory acidosis (2)

-In the setting of a combined metabolic and respiratory acidosis, correction of the respiratory acidosis component should be addressed prior to administration of bicarbonate or initiation of hemodialysis.-threatening acidosis. (2)

### References:

(1) Oxford Handbook of Critical Care 3<sup>rd</sup> edition

(2) Washington Manual of Critical Care

