Strategies for Electrolyte Replacement

Lauren N. Kuykendall MSN, RN, AGACNP-BC, CCRN

Objectives

- Identify when electrolyte replacement is needed and underlying cause
 - Potassium, Magnesium, Calcium, Phosphorous
- Understand different formulations, route, dosage and adverse effects of replacement
- Identify when goal replacement has been achieved
- Identify when maintenance therapy may be required

Hypokalemia

- Usually secondary to:
 - GI loss (vomiting, diarrhea)
 - Urinary losses (diuretics, RTA)

Also think about : co-existing electrolyte abnormality (hypomagnesemia), hyperaldosteronism, insulin therapy, albuterol, alkalosis)

- Indications for replacement:
 - Evidence of potassium loss
 - -Significant deficit in body potassium
 - Acute therapy in redistributive disorders (periodic paralysis, thyrotoxicosis)

Hypokalemia

- Symptoms: usually manifest when serum K <3.0
 - Muscle weakness (K <2.5), cramps, rhabdomyolysis
 - Respiratory muscle weakness
 - GI symptoms: anorexia, nausea, vomiting
 - Cardiac arrhythmias: atrial tachycardia, junctional tachycardia, AV block, ventricular tachycardia or fibrillation
 - EKG abnormalities: PAC, PVC, sinus bradycardia, ST segment depression, decreased amplitude of T-wave, increased amplitude of U-wave (mostly in V4-V6)
 - If prolonged hypokalemia: functional changes in the kidney and glucose intolerance

- Calculate potassium deficit (if normal distribution is presentdo NOT use in DKA or HONK)
 - Acute: .27meq/L decrease in serum K+ for every 100meq reduction in total potassium stores
 - Chronic: 1meq/L decrease in serum K+ for every 200-400meq reduction in total potassium stores

Simplified:

<u>Goal K – Serum K</u> x 100 = total meq K required serum Cr

10meq of KCL will raise the serum K by ~.1meq/L

Formulations

- Potassium Chloride : PREFERRED AGENT
 - Most patients with hypokalemia and acidosis are also chloride depleted
 - Raises serum potassium at a faster rate
 - Available as salt substitute, liquid, slow release tablet or capsule, and IV
- Potassium Bicarbonate/Citrate/Acetate:
 - can be used in patients with hypokalemia and metabolic acidosis
- Potassium Phosphate:
 - Rarely used (Fanconi syndrome with phosphate wasting)

Ongoing Losses

- In general, use oral therapy
- KCL with normal or elevated serum bicarb
- Potassium citrate/acetate/bicarbonate in presence of acidosis (diarrhea, RTA)
- no need for continued supplementation with chronic renal potassium wasting (potassium sparing diuretic is more effective):
 - Chronic diuretic therapy
 - Primary aldosteronism
 - Gitelman's, Bartter's syndrome

Adverse Effects

- Hyperkalemia
- Potassium is osmotically active- can increase tonicity of IV fluids
- Oral therapy- pills are large, can be difficult to swallow
- Peripheral IV Therapy:
 - Pain
 - Phlebitis

Make sure to recheck serum potassium 2-4 hours later to assess response to therapy

Goal of Therapy

- Prevent life threatening complications
- Urgency of replacement depends on severity, rate of decline and co-morbid conditions
 - Elderly
 - underlying heart disease
 - on digoxin or anti-arrhythmic drugs

Example

- 72 year old female admitted for weakness and dehydration due to acute gastroenteritis. She is having up to 6 BM/day. Her serum K on admission is 2.5 meq and serum Cr is 2.0. EKG reveals u-waves.
 - 1. How much potassium do you order?

<u>4-2.5</u> x 100 = 75meq

2

2. What formulation do you choose?

KCL; if bicarb is low then consider potassium bicarb or acetate

- 3. What route should the potassium be administered? 40meq (initial) oral and 40meq IV; (re-assess 2-4 hours later and give more orally if needed and tolerating po)
- 4. Serum potassium remains low, what else could be contributing?

Low magnesium, ongoing diarrhea

Hypomagnesemia

- Average daily intake: 360mg
- Presence of low magnesium (nearly 12% of hospitalized patients) suspected in following cases:
 - Chronic diarrhea
 - Hypocalcemia
 - Refractory hypokalemia
 - Ventricular arrhythmias
- Symptoms/Signs :
 - Tetany (seizures in children/neonates)
 - Hypokalemia
 - Hypoparathyroidism→ hypocalcemia (<1.2mg/dL)
 - Vitamin D deficiency (due to low calcitriol)
 - EKG changes: widened QRS, peaked T-waves, → dimunition, PR interval prolongation,
 - Ventricular arrhythmias (especially during ischemia or bypass), think TORSADES

- Goal of therapy:
 - maintain plasma magnesium concentration over 1.0mg/dL acutely in symptomatic patients
 - In cardiac patients, maintain Mg >1.7 (usually goal 2.0mg/dL) to avoid arrhythmias
 - Serum levels are poor reflection of actual body stores (mostly intracellular) so aim for high-normal serum level
- Avoid replacement in patients with reduced GFR
- Treat underlying disease (PPI, diuretics, alcohol, uncontrolled diabetes)
- Adverse effects:
 - Abrupt elevation of plasma Mg can remove the stimulus for Mg retention and lead to increased excretion
 - Diarrhea
 - Drug interactions
 - Magnesium intoxication, Aluminum intoxication

Hypocalcemia

- Clinical Manifestations:
 - Acute: serum Ca <7.5mg/dL
 - Neurologic: tetany (from paresthesias to seizures and bronchospasm)
 - Cardiac: prolonged QT, hypotension, heart failure, arrhythmia
 - Papilledema
 - Psychiatric manifestations
 - Chronic:
 - EPS, dementia, cataracts, dry skin
- Etiology:
 - Vitamin D
 - PTH
 - Hypomagnesemia
 - Drugs

- Correct for albumin
 - Ca lower by .8mg/dL for every 1g/dL reduction in serum albumin
 - or check ionized calcium
- Level can be altered by acid/base disturbance
- Add Vitamin D in following cases:
 - Hypoparathyroidism: Vitamin D (Calcitriol .25-.5mcg bid)
 - Vitamin D deficiency: 50,000IU/week for 6-8 weeks then 800-1000IU daily
 - Erogcalciferol (D3)
 - Cholecalciferol (D2)

- Goals of therapy:
 - Treat and prevent manifestations of hypocalcemia
 - In hypoparathyroidism: to raise serum Ca to low-normal range (8.0-8.5mg/dL)

Adverse Effects:

- Rapid infusion- bradycardia, hypotension
- Extravasation-tissue necrosis
- Hypercalcemia
- Hypercalciuria
- Constipation
- Hypophosphatemia
- Milk-alkali syndrome

Example

35 y/o male with hypoparathyroidism secondary to DiGeorge's presents with serum Ca of 6.2, albumin of 3.8, ionized Ca .77. Has some mild muscle cramps, otherwise asymptomatic.

- How do you initially treat his hypocalcemia?
 IV Calcium Gluconate 1g IV over 10-20min
- 2. Repeat serum Ca is 6.6, how do you proceed with treatment?

-start Calcium gluconate 1mg/mL in D5W 50mL/hr infusion

2. After initial treatment, what maintenance regimen should you initiate?

-Calcitriol (.5mcg bid, titrated up in this patient)

-Calcium carbonate (1950mg po tid in this patient)

Hypophosphatemia

- Due to:
 - Restribution
 - Decreased intestinal absorption (small bowel)
 - Increased urinary excretion
- Common situations:
 - Chronic alcoholism
 - IV hyperalimentation w/o phosphate supplementation
 - Refeeding syndrome
 - "Hungry Bone" syndrome
 - Respiratory alkalosis (hyperventilation)
 - Chronic ingestion of antacids (containing aluminum or Mg)
 - Hyperparathyroidism (primary or secondary)
 - Vitamin D deficiency
 - Fanconi syndrome (associated with multiple myeloma in adults)

Hypophosphatemia

Signs/Symptoms: <2.0mg/dL, severe usually when serum PO4
 <1.0mg/dL

Acute:

- Metabolic encephalopathy- irritability, paresthesias → confusion, seizure, coma
- Respiratory failure due to weakened diaphragm
- Reduction in cardiac output leading to heart failure
- Proximal myopathy, dysphagia, ileus
- Elevated CPK, rhabdomyolysis
- Coagulopathy with thrombocytopenia

Chronic:

- Hypercalciuria
- Increased bone resorption: Osteomalacia, Ricketts

Treatment

- Usually aimed at treating the underlying cause (resolution of diarrhea, Vit D therapy, d/c antacid, etc.)
- If tx is needed, oral therapy is preferred
 - Asymptomatic, serum PO4 <2.0mg/dL or symptomatic with serum PO4 1.0-1.9mg/dL
 - Available as tablet and powder/packets (sodium phosphate, potassium phosphate) 250-500mg tid-qid (w/ meals & HS) over 24 hours
 - Decrease dose by one-half in patients with reduced GFR
 - Increase dose in severely obese patients
 - Recheck after 12 hours to determine if additional/continued supplementation is required

Treatment

- Goal of therapy: increase serum PO4 to 2.0mg/dL
- Side effects of therapy:

-Oral: Diarrhea, nausea, hyperkalemia (K-phos)

- -IV: Hyperphosphatemia \rightarrow hypocalcemia, AKI, arrhythmia
- Maintenance therapy is not usually required