Fluids and Electrolytes

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What We Will Discuss

- Major electrolytes, too low, too high and what to do
- Common fluid and electrolyte issues that we see everyday in the PICU
- And of course lots multiple choice questions

What We Will Not Discuss

- TPN
- Any tubules, glomeruli, or arterioles
- Ca binding proteins
- Na-K transporters
- Dialysis, apart from specific indications

First - Na

- 90% extracellular
- Major determinant of extracellular osmolality
- Very important for CNS
- Large rapid changes can be life threatening
- Small changes are harmless but warn of other processes
- Check electrolytes!

Hyponatremia - causes

- Decreased Na – increased loses, AI, CSW, diuretics, osmotic loses (DKA)
- Increased Na – free water retention exceeds Na retention, CHF, cirrhosis, nephrotic syndrome, renal failure
- Normal Na - SIADH

Which of the following drugs is NOT associated with SIADH

A. Vincristine
B. Haldol
C. Azithromycin
D. Ecstasy
E. SSRI

C. Azithromycin

Hyponatremia: Symptoms & Treatment

- Cellular swelling and cerebral edema
- Lethargy, N/V, cramps, confusions
- Seizures and coma <120 (acute)
- Chronically CNS cells compensate – rapid correction - osmotic demyelination
- Treatment – 1st stop herniation/seizures
- 5-6 ml/kg 3% will raise Na 5mEq/L
- Chronic or acute with CNS sx – 0.5mEq/L/h
- Acute with no CNS sx – 0.7-1mEq/L/h
- MUST follow levels!

How many mEq of Na are in a L of 3% Saline

A. 513

B. 462

C. 300

D. A lot

E. Too much math

Answer A. Normal saline is 0.9% 154mEq/L. Divide by 9. Add to 154 to get “1% saline”. Multiple by 3. Other options include google. Note “normal saline” is actually not.

Hypernatremia: Causes

- Decreased Na – free water loses > Na loses, diarrhea, iatrogenic from insufficient free water, diuresis
- Normal Na – DI
- Increased Na – usually iatrogenic – 3% in TBI, $NaHCO_3$ during resuscitation, improperly prepared infant formula

Hypernatremia: Symptoms & Treatment

- Increased osmolality, most issues in CNS
- Irritability, spasticity, N/V, seizures, coma and of course death
- Decreased brain cell volume – tearing of vessels, subcortical or subdural bleeds, vascular congestion, CVT, demyelination
- Accumulation of idiogenic osm in CNS cells occurs with time
- Rapid correction – brain edema
- Correct over 48 h no faster than 1 mEq/L/H

Your otherwise stable TBI patient is on 3% saline gtt. Na is 156. Labs show non-gap metabolic acidosis. WTF?

A. Shock
B. Hyperchloremia
C. Hyperphosphatemia
D. New onset DKA
E. Salicylate abuse

Answer: B in setting of hyperchloremia, kidneys waste bicarb to maintain electro-neutrality.

Now, K^+

- Mostly intracellular
- Hypokalemia is common, rarely fatal
- Hyperkalemia is uncommon and very bad
- Mostly K is managed by kidneys and GI tract
- Also affected by acid-base balance, insulin, catecholamines, Mg and aldosterone
- Kidneys secrete K during alkalosis and resorb it during acidosis
- Cells exchange K^+ for H^+ when acidosis is caused by excess H^+ therefore….

Hyperkalemia seen with DKA is due to:

A. Inappropriate fluids in the peds ED and PICU
B. Insulin deficiency
C. Excess H^+ ions
D. Organic acids
E. Everyone knows hyperkalemia with DKA isn't really real, I am SO much smarter than this question

Answer: B because the acidosis is caused by organic acids, not H^+ ions, K^+ does not leave the cells to maintain electroneutrality, it leaves because of insulin deficiency. Simplified, of course.

Hypokalemia: causes, symptoms & treatment

- Beta-agonists, hyperaldosteronism, elevated renin, diuretics, osmotic diuresis, GI loses, malnutrition, re-feeding, geophagia, Barium poisoning, Barter syndrome, RTA, drugs…
- Symptoms – flattened T-waves, ST depression, U-waves, arrhythmias, weakness, ileus
- Treat – oral 1 mEq/kg or IV 0.5mEq/kg
- "Potential for catastrophic drug error in potassium replacement is real."
- Ask – does this K^+ *really* need to be replaced?

Hyperkalemia: Causes, symptoms & Treatment

- Causes – redistribution, administration error, blood products, rhabdo, hemolysis, renal failure, TLS, metabolic acidosis, AI
- EKG – peaked T-waves, decreased P and R wave, widened QRS, bradycardia, classic sine wave blending P and QRS complex
- EKG can progress over minutes, CPA, V-fib/tach can happen at any point in this progress
- < 6.5 remove K^{+} +/- kayexalate and monitor
- >6.5 or EKG changes, Ca^{+2} , insulin/glucose, sodium bicarb, albuterol, dialysis, loop/thiazides diuretics

You are NF senior. A pt has a K^+ of 7.5 with EKG changes. What is the 1st thing you should do?

A. Order calcium
B. Order insulin/glucose
C. Order sodium bicarb
D. Call rapid response
E. Call code blue
F. Call PICU attending

Answer: Discuss. Real life is not multiple choice….

Same patient has pulseless v-tach, the first thing you should do?

A. CPR
B. Defibrillate
C. Calcium
D. Sodium bicarb
E. Insulin
F. Call a code blue

Answer: start (or make sure someone else starts) CPR. Everything else should happen simultaneously, again really life not multiple choice.

Hypomagnesemia

- Mostly intracellular, muscle and bone
- Dietary deficiencies, malabsorption, renal dz
- Drugs – tacrolimus, cyclosporin, amphotercin, cisplatin and diuretics
- Seizures, hypertension, ventricular arrhythmias, coronary spasm and NM “stuff”
- Treat with Mg – 25-50 mg/kg watch for respiratory depression and hypotension

Hypermagnesemia

- Causes- iatrogenic and renal failure
- Symptoms – pseudocoma, hypotension and respiratory depression, arrhythmias (theme), decreased DTRs, eventually flaccid quadriplegia
- Treatment – calcium (direct antagonist), lasix, dialysis

You are the PICU fellow called to RRT for a 15 y/o seizing, unresponsive, hypertensive small bowel transplant patient. You treat the seizure, secure the airway, transfer the patient and order labs. You expect…

A. High Mg, low tacrolimus
B. Low Mg, high tacrolimus
C. High Mg, high tacrolimus
D. Low Mg, low tacrolimus.

Answer: B. Both low Mg and high Tac will lower seizure threshold and will also do so synergistically.

Hypophospatemia

- Mostly in bones, normal levels vary with age, less is normal with age
- Symptoms only when < 1.5 – no ATP, 2,3-DPG
- Refeeding syndrome, burns, DKA, respiratory alkolosis, and deficient TPN
- Symptoms – weakness, respiratory depression, decreased O2 delievery
- Treat….with phos!

Hyperphosphatemia

- Causes – TLS, renal failure, iatrogenic
- Symptoms – hypocalcemia, seizures, cardiac arrest
- Treat – fluids, calcium, mannitol, dialysis

Anorexia, DKA, renal failure and new ALL can all disturb phos, in which directions?

A. Re-feeding low phos, DKA high phos, renal failure low phos, TLS high phos
B. Re-feeding high phos, DKA low phos, renal failure high phos and TLS low phos
C. Re-feeding high phos, DKA high phos, renal failure low phos and TLS high phos
D. Re-feeding low phos, DKA low phos, renal failure high phos and TLS high phos

Answer: D. Note re-feeding syndrome and TLS are opposite (K^+ too). DKA pee out too much, renal failure pee out not enough.

Hypocalcemia

- Causes – reduced PTH, vitamin D, alkolosis, hyperphos, drugs/toxins, TLS, blood products (why?), the vague but common“critical illness”
- Symptoms – decreased muscle contractions, stridor, apnea, tetany, seizures, muscle spasms, hypotension, CHF, arrythmias, prolonged QT
- Treat – calcium. $CaCl_2$ only via CVL, calcium gluconate can go in PIV.

Hypercalcemia

- Causes – hyper-PTH, vitamin D toxicity, malignancy, immobility, thiazides
- Symptoms – hypertension, constipation, abdominal pain, polyuria, dehydration, stones, hypotonia, shortened QT, arrhythmias
- Remember stones, groans, bones and psychiatric moans from Step 1?
- Treat – hydration, lasix, calcitonin (“tones” down calcium), bisphosphonates
- Note – with calcium lasix and thiazides have opposite effects.

ICU FEN Issues: How Much?

- 4:2:1 rule generally applied
- Vented patients need less, don't lose H_2O from respiratory tract – 2/3 to 3/4 MIVF
- Rhabdomyolysis, DKA need more 1.5MIVF
- Shock, on-going loses – replace with boluses as needed or else fluid overload can easily occur
- Septic shock should not be treated with 1.5 MIVF instead of or even in addition to boluses, you risk fluid overload, high Na, Cl and glucose and you won't find this in ANY septic shock algorithms, go ahead check your PALS card.

ICU FEN issues: what?

- D5NS +/- KCl is safe go-to fluid
- Post-operative patients are at risk for hyponatremia – should get NS
- Any neuro pt should absolutely get NS
- 1/2NS – small infants, DKA, borderline high Na
- 1/4NS – only with hypernatremia

PICU FEN Issues – when to replace electrolytes?

Probably let it be if some or all of below is true

- Stable patient
- Moderate deficiency (K or phos of 3.3)
- Eating patient
- No expectation of on-going loses
- Deficit not consistent with clinical picture - suggesting transient or spurious value

Probably need to replace if ANY of below is true

- Symptomatic or critical
- Severely low levels (Mg is 1)
- NPO on IVF
- Expectation of on-going loses (giving more lasix)
- Deficit is consistent with clinical situation
- Special cases – low Mg in transplants, low Na in TBI

ICU fluid issues: what?

- TBI – no glucose for 72 h
- Everyone else D5
- Small infants or anyone at risk for hypoglycemia, D10

ICU FEN issues: checking labs

- Most ICU patients need lytes checked at or near admission, especially critical patients, asthmatics, DKA
- Who needs daily lytes? General guidelines…
- Anyone NPO/IVF
- Anyone on TPN that is being actively titrated
- Anyone on diuretics being actively titrated
- Anyone severely critical

PICU FEN Issues: My patient is not peeing…fluids or lasix

Lasix

- No evidence of shock
- Good perfusion
- Not tachycardic
- Normo/hypertensive
- CVP > 6-8
- BUN and Cr normal
- Suspicion of fluid overload (wet CXR, edema)
- Clinical explanation for urinary retention (PCA)

Fluid

- Evidence of shock
- Tachycardia
- Hypotension
- Dry mucus membranes, sunken fontanelle
- Low CVP
- Elevated BUN
- Clinical explanation for fluid deficit (GI loses, OR)

I am still not sure – insert foley