Electrolyte Disturbances

...And a Touch of Physiology

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BCRenal

CKD EDUCATION
· ONLINE CME FOR PRIMARY CARE ·

Webinar Logistics

Feel free to ask questions in chat (I have been told these can be private to me only); will try to address by section or at the end.

Feel free to email questions / feedback to my email.

Disclosures

No relationships with commercial interests.

No known or potential conflicts of interests.

Objectives

1) Common laboratory abnormalities
2) Practical outpatient approaches to managing electrolyte abnormalities
3) “Uremia” - an abnormal electrolyte?
4) Dialysis indications & Nephrology referral
5) Renal autoregulation & induced impairment
6) Specific entities: CHF and AKI vs. CKD

Common Laboratory Abnormalities

Electrolyte challenges: Na

Hypernatremia

Hypernatremia

Reflection of cellular dehydration and lack of free water (relative to solute):

- Daily obligate losses from breathing, sweating, GI, GU

*~1000mL not account for metabolic H2O generation

Free water losses

Pure free water intake deficit

Sodium overload / ingestion

Hypernatremia

Free water losses:

- Renal concentration defect (osmotic diuresis, diabetes insipidus, diuretics - loop > thiazide)
- GI (diarrheal losses)
- Insensible / sweat (burns)

Give free water!

Stop diuretic? (depends on indication)

Uosm < Sosm = diabetes insipidus (central vs. nephrogenic)

Check serum Glc

Hypernatremia

Intake deficit:

- Inability to drink
- **Limited access to water**
- Impaired thirst mechanism (thirst stimulated by increase in osmolality - osmoreceptors in brain)

Give free water!

*often times, you don't see significant hyperNa in outPts because thirst is very powerful driver (even in diabetes insipidus - 10-15L urine output/day)

Hypernatremia

Sodium overload / ingestion:

- Iatrogenic (3% NS, Na tablets, hypertonic irrigation)
- Salt water / soy sauce
- Primary hyperaldosteronism
- Cushing’s syndrome

Give free water!

Limit sodium intake

Consider endocrinological etiology (*rare)

Hypernatremia

Practically speaking, seen most often in:

- post-ATN/obstructive diuresis
- aggressive (induced) diuresis
- critical illness
- terminal course / end of life

***Give free water!**

#ProTip: Assuming an average 70kg male, every 10 mmol/L increase in Na corresponds to approximately a 3L free water deficit

^caveat: assumes your patient is a bath tub

*Unless your patient is end-of-life; then hypernatremia is part of the expected course

Electrolyte challenges: Na

Hyponatremia

Hyponatremia

THE Most common electrolyte problem

- If severe: Cerebral edema
- If chronic: Associated with increased risk of falls + osteoporosis

Cerebral Edema -

neurologic symptoms

(typically <125 mmol/L)

- Nausea
- Confusion
- Headache
- Yawning / lethargy

Hyponatremia

THE Most common electrolyte problem

- If severe: Cerebral edema

  ***these more severe symptoms would prompt more acute management**

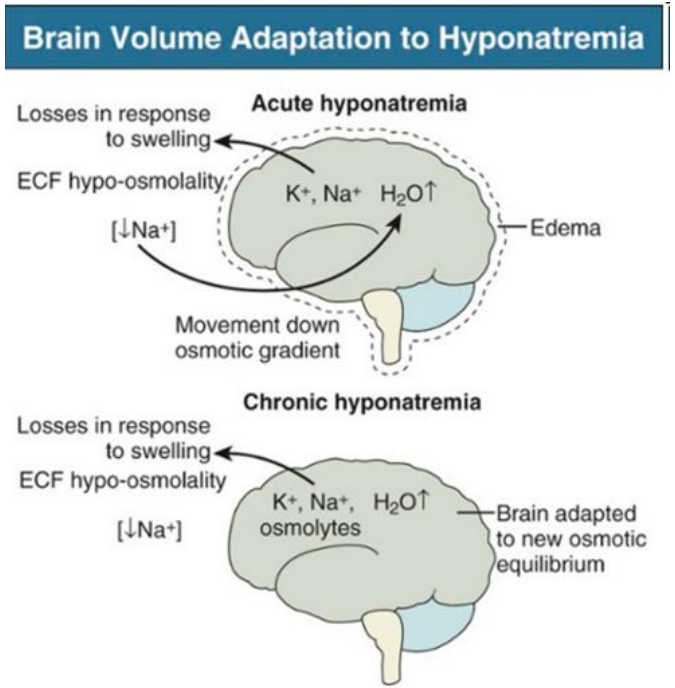
Cerebral Edema - neurologic symptoms

(typically <125 mmol/L)

- Vomiting
- Reversible ataxia
- Psychosis / disorientation
- Seizures
- Coma

Hyponatremia

Physiologic adaptations



Clinically: how to act?

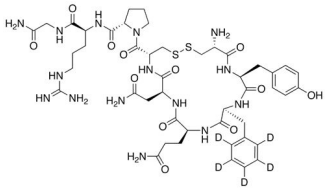
- Symptomatic
  - refer for acute care
- Asymptomatic

Hyponatremia

First steps:

- Where is the H2O?

- Where is the ADH?



- Typically, excess free water ➔ fluid restrict

- Stop thiazide medications (interfere with formation of dilute urine - i.e. cannot get rid of free water)

Hyponatremia

First steps:

- Where is the H2O?

Endogenous:

- *approximately 0.5-1L “generated” in daily metabolism

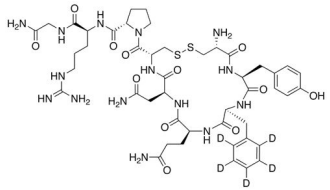
Exogenous

- PO intake
- IV fluids / meds

Hyponatremia

First steps:

- Where is the ADH?



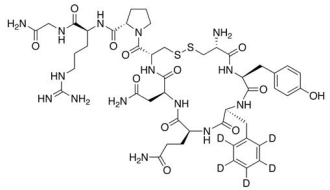
Osmotic stimuli

- Changes as small as 1% in sOsm cause release of vasopressin/ADH

Hyponatremia

First steps:

- Where is the ADH?



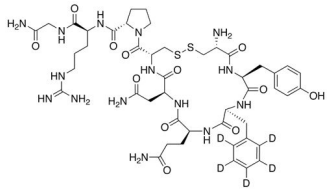
Non-osmotic stimuli

- Decreased effective circulating volume (heart failure, cirrhosis, GI losses)
- ➜ parasympathetic stimulus for ADH release

Hyponatremia

First steps:

- Where is the ADH?



Non-osmotic stimuli

- Nausea, post-operative pain, pregnancy
- *Hypovolemia -- typically needs large (~7%) decrease in blood volume before response*

Hyponatremia

First tests:

- Repeat Na, K, Glc, BUN, Cr
- (Serum osmolality)
- Urine osmolality
- *Urine: Na, Cl
- UA for SG

Remember:

- Typically, excess free water ➔ fluid restrict

- *Stop thiazide medications (interfere with formation of dilute urine - i.e. cannot get rid of free water)

Hyponatremia

First tests:

-
-
-
-
-
- UA for SG

#ProTip: UA SG vs. Uosm

- 1.000 - 0
- 1.010 - 350 → “concentrated urine”
- 1.020 - 700
- etc.

Hyponatremia

What the tests mean:

- uOSM vs. sOSM (or calc: sOSM: 2xNa + BUN + Glc)
  - Concentrated urine (uOSM approx >300)
  - Dilute urine
- uNa and uCl low

Common things:

- Thiazide Diuretic
- Physiological stress (pain, anxiety, nausea)
- “Pre-renal”
  - CHF / hepatic / nephrotic
  - True hypovolemia
- Low solute diet

When you see a salt problem: think **water**.

Electrolyte challenges: K

Hyperkalemia

Hyperkalemia

Balance of intake and excretion (renal 90-95% / GI 5-10%):

- normalizing bowel care is paramount!
- **stop offending medications** (K-sparing diuretics, Septra)

Potassium-binders:

- Sodium polystyrene sulfonate
- Calcium polystyrene sulfonate

Coming soon:

- Patiromer (FDA Oct 2015, Health Canada Oct 2018)
- Sodium zirconium cyclosilate (FDA May 2018, Health Canada - in review)

Hyperkalemia

Balance of intake and excretion (renal 90-95% / GI 5-10%):

- Low K intake until renal / GI systems normalized

High Potassium foods:

- Tomatoes
- Potatoes (chips)
- Fruits: bananas, oranges, watermelon
- Spinach, broccoli, beans
- avocados

“You aren’t what you eat - **you are what you don’t poop**.”

Electrolyte challenges: Ur

“Uremia”

Urea

Urea by itself, not a toxic entity; however, used as a surrogate measure of renal function / clearance ∴ high urea ≠ uremia

Uremia: symptoms associated with (toxic) metabolite accumulation in renal impairment

- Mental status changes / visual changes / fatigue
- Anorexia / taste changes / nausea / vomiting / weight loss
- Itch / restless legs / cramps

When to Dialyze

*ahem, call Nephrology

Indications for Dialysis

Refractory:

- Threatening electrolyte disturbances (K+)
- Acid-base stabilization
- Volume management
- Dialyzable intoxications
- “Uremia”

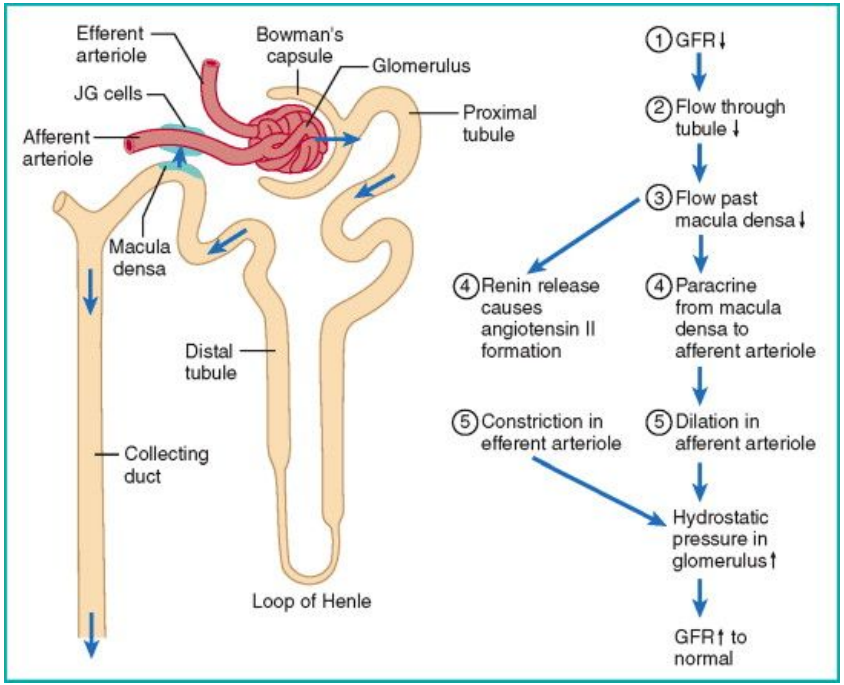
Often times we do not jump first to dialysis, but institute medical management; however - red flags:

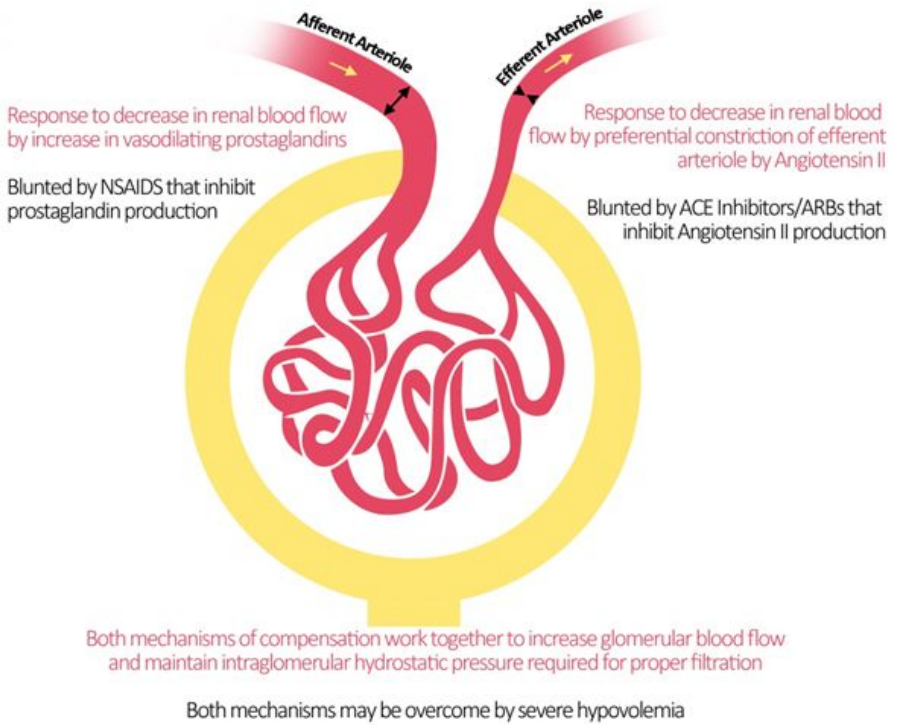
- **Lack of urine output**
- **End organ impairment: neurological, respiratory, cardiac**

**when to call us? Whenever you’re not sure or when you’re worried!*

Medication Considerations

Renal Autoregulation





Renal Autoregulation

Renal Tri-fecta of Doom

ACEi / ARB

NSAIDs

(Diuretics)

The kidneys have a **native ability** to take care of themselves.

Balancing CHF and CKD: heart or kidney?

Principles in CHF

Focus on the goal

Effective Diuretic Dose

Sacrificing GFR

Effusions

Subjective and Objective Assessment

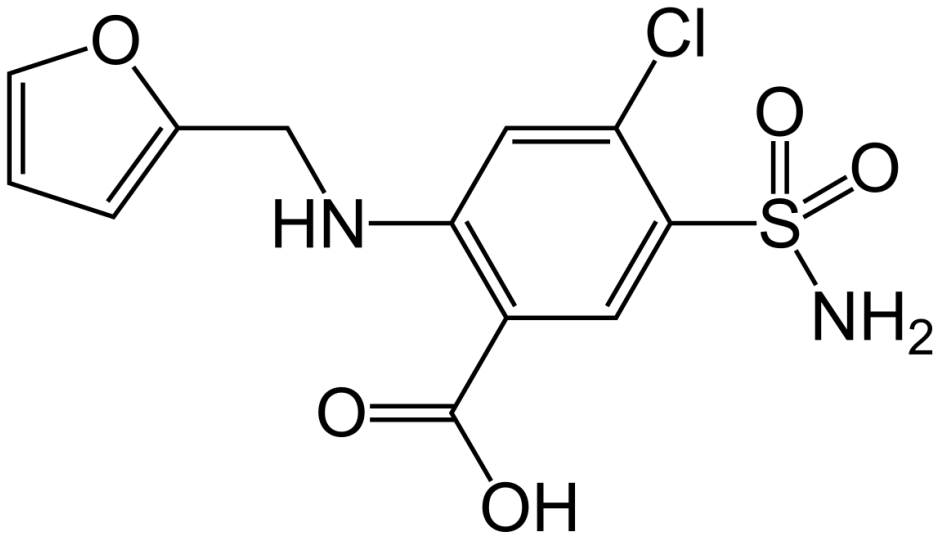
Refill Rate

Effective Diuretic Dose

Confounded by:

- GFR
- Albumin
- GI absorption (10-100% furosemide bioavailability)

*effective dose = dose that produces prompt urinary response (within 1-2 hours)



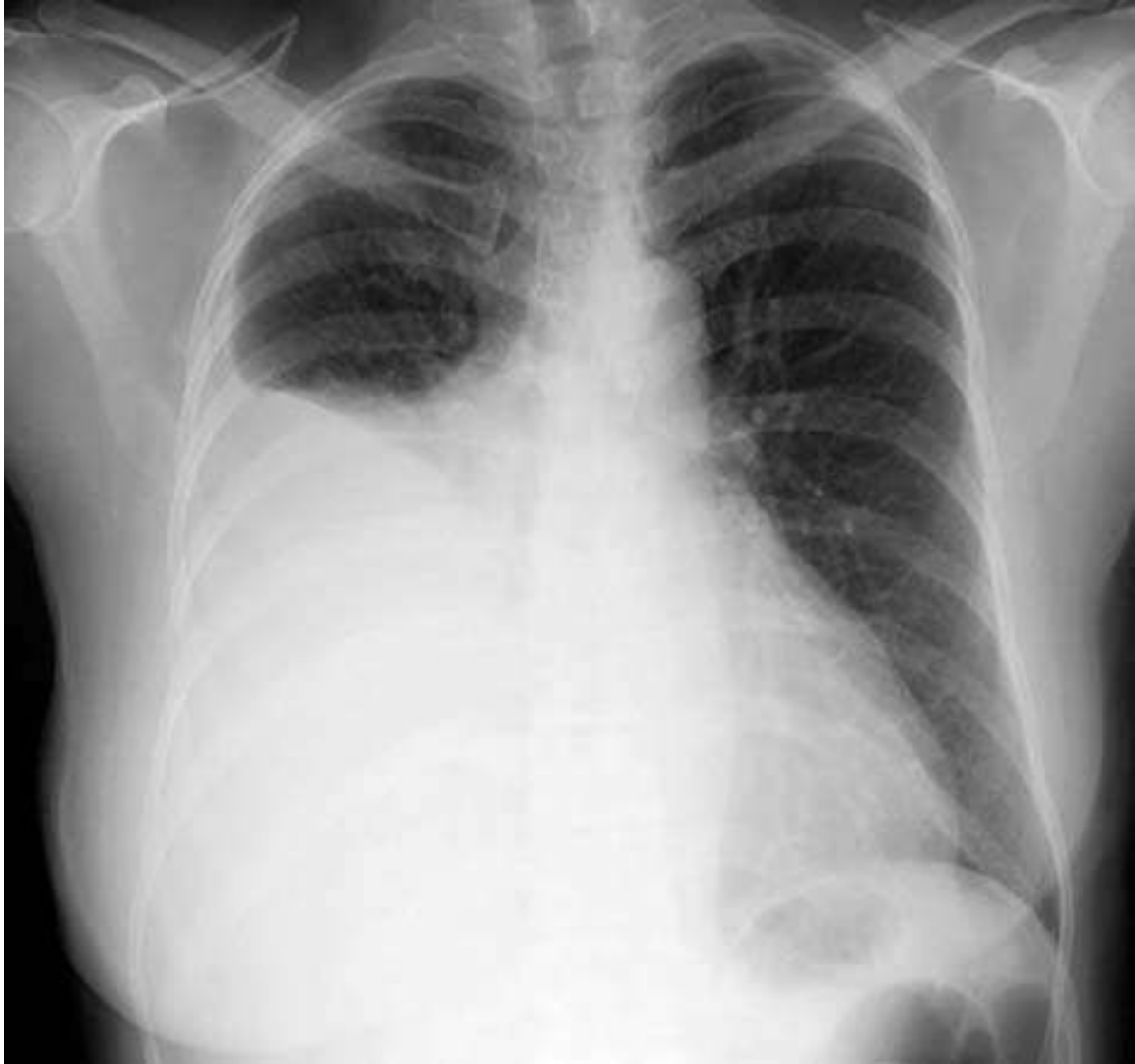
Sacrificing GFR

Furosemide by itself *does not* cause AKI or hypotension - *MUST* be accompanied by intravascular depletion ∴ no urine output (weight loss), no AKI

*However, in principle, you are trying to shift intravascular volume to promote re-recruitment of extravascular (interstitial) fluid.

Fussy Effusions

Re-recruitment of effusions ***takes time***; if acutely symptomatic, may need to have therapeutic drainage - otherwise plan to wait weeks for resolution/resorption.



Evaluation & Refill Rate

Focus on:

- Symptomatic improvement (ambulation distance, orthopnea, sleep, coughing, tireness)
- Controlled Objective verification (weight, waist/leg circumference, O2 requirements)

Remember: progress, not perfection

A 0.5-1kg decrease per day is plenty

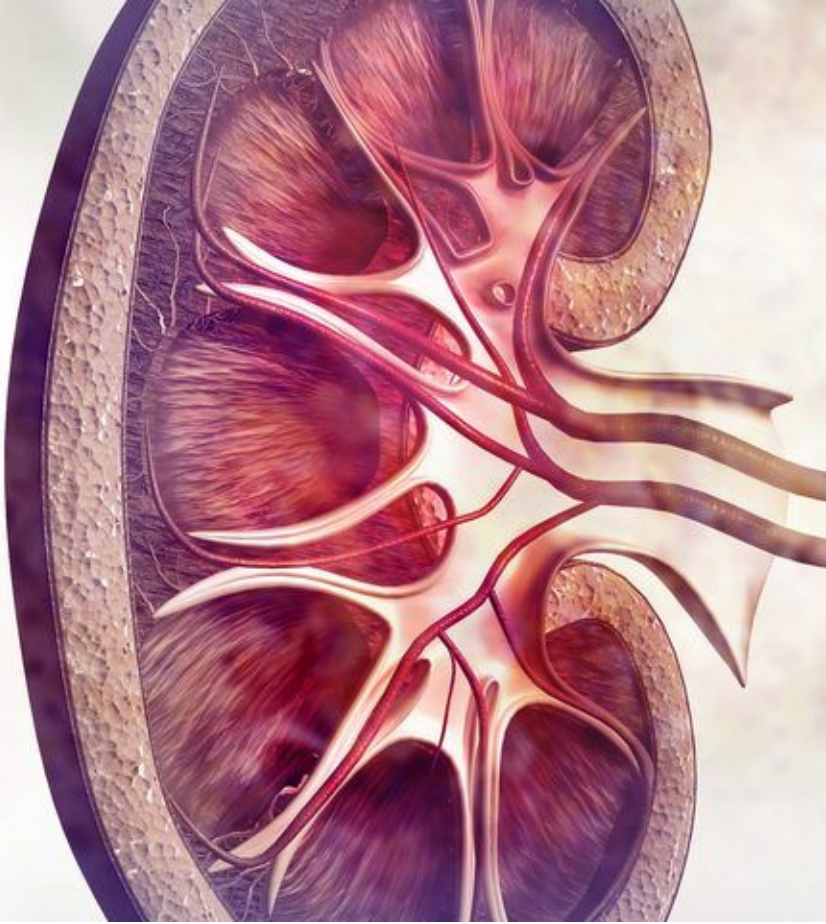
Take Home Message(s)

- Think: water
- Trust in renal autoregulation
- Medication ‘sick day’ action plan
- Reassess diuretic use
- Sometimes you will need to sacrifice GFR for volume status - and that’s okay

Questions?

Clinical cases / conundrums?

Thoughts



Thanks!