APPROACH TO ELECTROLYTE DISORDERS IN LTC

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PRESENTER DISCLOSURE

No conflicts of interest to disclose

LEARNING OBJECTIVES

1. Approach to workup and management of sodium disorders
2. Approach to workup and management of potassium disorders
3. Approach to abnormalities in extended electrolytes
4. What to know about LTC patients who are receiving dialysis

LEARNING OBJECTIVES

1. **Approach to workup and management of sodium disorders**
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SODIUM DISORDERS

- <u>Keypoints:</u>
- Sodium abnormalities are really due to **changes in WATER balance**
- Both water and sodium balance is handled by the kidneys
  - Water handling via aquaporin channels (as directed by ADH)
  - Sodium handling via various transporters (and affected by aldosterone)
- **Hypo**natremia = relative **water excess**
- **Hyper**natremia = relative **water deficit**

HYPONATREMIA

- **Hypo**natremia = relative **water excess**

What we teach the residents:

- 1. Send serum osmolarity
- 2. Send urine osmolarity
- 3. Send urine sodium & look at volume status

HYPONATREMIA

1. Send serum osmolarity:

- **Hyperosmolar** hyponatremia
  - Hyperglycemia – can correct as follows: for every 10 points glucose above 10 correct sodium UP by 3
  - Ie. Na 129 with gluc 30: 20 above 10 → 2 x 3 = 6 → corrected Na is 135
- **Iso-osmolar** hyponatremia "pseudo-hyponatremia"
  - Laboratory artifact due to paraprotein or high immunoglobulins
  - Whole blood sodium will be more accurate
- **Hypo-osmolar** hyponatremia *what people usually mean by hyponatremia*

HYPONATREMIA

2. Send urine osmolarity:

- **This looks at ADH activity**, ie. Is ADH active? And is that appropriate?
  - If ADH is on → water is being reabsorbed → urine osmolarity is HIGH

- Various cutoffs exist for what is HIGH vs. LOW

HYPONATREMIA

2. Send urine osmolarity:

- Comparison with serum osmolarity:

Urine osm < Serum osm	Urine osm < 100	**ADH is OFF!** Primary polydipsia Low solute intake “tea-and-toast” “beer potomania”
Urine osm < Serum osm	Urine osm > 100	Grey zone…
Urine osm > Serum osm	Urine osm > 100	**ADH is ON!** Check urine Na

HYPONATREMIA

3. Send urine sodium:

- **This looks at sodium handling**, ie. Are the kidneys trying to reabsorption sodium?
  - If kidneys perceive volume depletion → reabsorb sodium → urine Na is LOW
  - This can help distinguish between intravascular depletion as perceived by kidneys vs. inappropriate ADH activity despite no need for water reabsorption

- Note: urine sodium can be confounded by some things – not interpretable
  - Patient is on diuretic
  - Patient has just received IV fluids

HYPONATREMIA

3. Send urine sodium:

- If ADH is already on:

Urine osm > Serum osm Urine osm > 100 "ADH is ON"	Urine Na < 30 "Na is being reabsorbed"	**Intravascularly deplete!** Volume depletion Heart failure Cirrhosis Third spacing
Urine osm > Serum osm Urine osm > 100 "ADH is ON"	Urine Na > 30 "Na is NOT being reabsorbed"	**ADH is inappropriately on!** SIADH Misc* causes

HYPONATREMIA

SIADH

= Syndrome of Inappropriate ADH

- Physiologic causes:
  pain, stress, nausea
- Medication related causes:
  antidepressants, antipsychotics, antiepileptics
- CNS: intracranial process
- Respiratory: pneumonia, malignancy

Misc* causes

= looks like SIADH

- Adrenal insufficiency
- Hypothyroidism

HYPONATREMIA

How about a practical approach to LTC patients?

- First correct for hyperglycemia – it may end up being normal
- Look for medication culprits:
  - Are they on diuretics? (nidus for volume depletion, plus you know urine Na not useful)
  - Are they on antidepressant/antipsychotic/anticonvulsants? (possible SIADH)
- Assess their volume status:
  - Are they clearly dry or clearly overloaded?
- If nothing obvious – send serum osmolarity, urine osmolarity +/- urine sodium, TSH

HYPONATREMIA

What do you do while the labs are in process?

- If they are clearly volume deplete – stop diuretics and increase intake + hydration
- If they are clearly overloaded and need diuresis anyway – do it
- If you aren't sure – employ fluid restriction (less than 1.5 L daily to start)
  - This addresses polydipsia, low solute intake, SIADH, and helps with reduced ECFV
- Repeat labwork to ensure trending in the right way

HYPONATREMIA

What if you arrive at a conclusion of SIADH?

- Is there a physiologic reason?
- Are there offending medications? Consider holding +/- consult with psychiatry/neurology
- Do they have localizing respiratory symptoms? Consider chest imaging → treat
- Enforce further fluid restriction – go from 1.5 to 1 L per day if needed
- If they are not overloaded – add salt tablets (1g TID to start)
  - This may also help with those with poor solute intake

HYPONATREMIA

When do you send them to acute care?

(presuming this is within GOC)

- Symptomatic hyponatremia – poor LOC, seizures, refractory nausea/vomiting
- Severe hyponatremia – sodium less than 120
- Worsening despite initial measures – do they need IV fluids? IV diuresis? 3% saline?
- They need acute care for other reasons – heart failure, decompensated cirrhosis, sepsis

HYPERNATREMIA

- **Hyper**natremia = relative **water deficit**

A lot easier than hyponatremia, either:

- Patient has **poor access to free water** or losing too much free water
- They have developed **diabetes insipidus**
- (getting too much hypertonic fluids, unlikely in LTC setting)

HYPERNATREMIA

- Hypernatremia is typically not seen in cognitively intact, ambulatory patients
  - Thirst is very powerful driver against water deficit!
- **LTC patient population especially prone to hypernatremia**
- First look at medications:
  - Diuretics causing water deficit
  - Medications resulting in poor LOC or oral intake
- Increase hydration wherever possible
  - If tube fed → increase free water flushes
- Hypernatremia typically a **harbinger of dying process**

HYPERNATREMIA

- Quick word on diabetes insipidus
  - Central DI: hypothalamic-pituitary defect resulting in reduced ADH secretion
  - Nephrogenic DI: reduced response to ADH in the distal collecting tubules
- LTC patients with history of lithium use can be at risk of nephrogenic DI
  - Features: polydipsia, polyuria
  - Refer to nephrology if suspected

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2. **Approach to workup and management of potassium disorders**
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POTASSIUM DISORDERS

- <u>Keypoints:</u>
- Potassium is an **intracellular** cation
  - ie. What you measure in the serum is only a fraction of the patient's potassium stores
- Potassium is **obtained** via GI absorption
- Potassium is **excreted** via kidneys
  - As long as there is reasonable renal function and urine output → no problem!
- Potassium is subject to various mechanisms for **shifting** in and out of cells

HYPERKALEMIA

- When potassium is elevated:
- Potassium is an **intracellular** cation
  - Is this real? Could it be a hemolyzed sample
- Potassium is **obtained** via GI absorption
  - The potassium has to be coming from somewhere → dietary modification +/- binder use
- Potassium is **excreted** via kidneys
  - Could something be impairing renal function or renal clearance of potassium?
- Potassium is subject to various mechanisms for **shifting** in and out of cells
  - Is there an acidotic state? Or hyperglycemic state?

HYPERKALEMIA

Common culprits for new hyperkalemia:

- Spurious result due to hemolyzed sample
- **Medications, medications, medications**!
  - MRAs – spironolactone, eplerenone, finerenone
  - ACEi, ARB, ARNI
  - Septra
- Dietary indiscretion
- Acute kidney injury

HYPERKALEMIA

What do you do?

- Repeat labdraw if markedly different from previous or ‘hemolysis’ described
  - To minimize hemolysis, refrain from tourniquet use and repeated fist clenching
- **Review medications and stop offending agents**
- Order renal or potassium limited diet
- Consider binder use (SPS, calcium resonium) – either acutely or chronically
- If accompanying renal injury → address underlying cause

HYPERKALEMIA

When do you send them to acute care?

(presuming this is within GOC)

- Potassium greater than 6
- Associated cardiac instability – specifically bradyarrhythmia
- Accompanying acute kidney injury or related acute illness requiring acute care

HYPOKALEMIA

- <u>When potassium is low:</u>
- Potassium is an **intracellular** cation
  - ie. It generally takes more than you think to replete their total body potassium
- Potassium is **obtained** via GI absorption
  - Are they having diarrhea, poor intake, or other GI losses?
- Potassium is **excreted** via kidneys
  - Are they being diuresed?
- Potassium is subject to various mechanisms for **shifting** in and out of cells
  - Check magnesium and replete if low

HYPOKALEMIA

What do you do?

- Replete enterally
- Address cause for GI losses or poor intake
  - Remember to look for stool softeners or prokinetics
- Stop diuretics
  - Specifically thiazides, loop diuretics
- Replete magnesium if low

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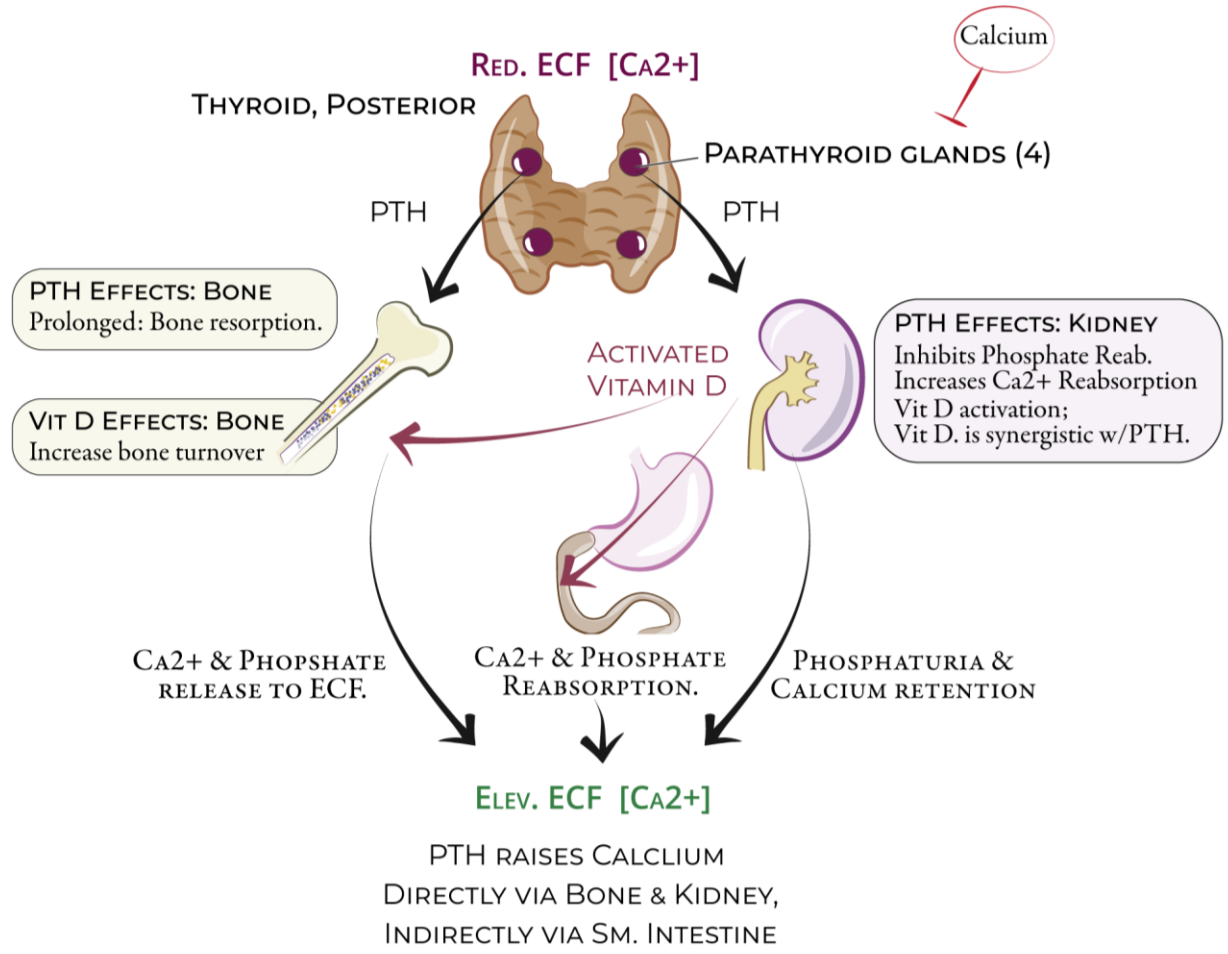
CALCIUM & PHOSPHATE REGULATION

Kidney	Excretes Ca, P Responds to PTH Activates 1,25 Vit D
Parathyroid	Secretes PTH Response to Ca, P
Bone	Releases Ca, P Responds to PTH Responds to 1,25 Vit D
GI tract	Reabsorbs Ca, P Responds to 1,25 Vit D
Skin	Activates 25 Vit D



HYPERPARATHYROIDISM

	Calcium	Phosphate	PTH	
Primary	↑	↓	↑	Driven by autonomous PTH secretion Results in overall Ca absorption & bone release Results in overall P renal excretion Treatment: parathyroidectomy
Secondary	↓	↑	↑	Driven by P accumulation, low Vit D activation “compensatory hyperparathyroidism” Most common scenario in CKD patients Treatment: P binder, 1,25 Vit D, dialysis
Tertiary	↑	↑	↑	After prolonged 2’ hyperPTH → gland now autonomous Treatment: cinacalcet, parathyroidectomy

HYPERCALCEMIA

- If mildly elevated, **repeat with ionized calcium**
- If **PTH** is high → primary or tertiary hyperparathyroidism → Endo +/- Nephrology
- If PTH normal/low, think about:
  - **Medications**: calcium, vitamin D, thiazides, lithium → stop if able to and then repeat
  - Malignancy: myeloma, bone metastases, lymphoproliferative, PTHrP from solid organ CA
  - Granulomatous processes: sarcoidosis, TB
  - **Immobility**: diagnosis of exclusion → but more common in LTC!
- If serum Ca > 3 or symptomatic → send to acute care (if within GOC)

HYPERPHOSPHATEMIA

- High phosphate – mostly seen in renal impairment
  - Due to inability to excrete phosphate, compounded by:
  - Reduced 1,25 vitamin D activation & increased bone turnover in response to PTH
- Clinical significance?
  - Observational & animal models suggest increased cardiovascular risk
  - Symptom burden from ++pruritus
  - Not intrinsically nephrotoxic

HYPERPHOSPHATEMIA

Management:

- Dietary – dietary phosphate restriction
  - But can be difficult in LTC setting or due to cognitive impairment
- Binder therapy – calcium vs. non-calcium based binders
  - Hence why patients are prescribed Tums to be taken with meals
- Clearance – optimizing dialysis

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HEMODIALYSIS

Logistics:

- 3 main in-centre HD units: RCH, SMH, ARH
  - LTC patients typically not candidate for community dialysis units
- Patients generally are on 3x/week schedule: M/W/F, T/Th/Sat or Sun
  - 3 shifts in a day – morning, afternoon, evening
- Nephrologists round on every shift for in-centre HD patients
  - Daytime coverage is provided by same nephrologist – rotate weekly
  - Evening and weekend coverage is provided by on-call nephrologist
  - All patients retain a ‘primary’ nephrologist for overall direction of care & consistency

HEMODIALYSIS

What can we do on HD?

- Electrolyte management, ie. clearance
- Volume management, ie. ultrafiltration
- Blood pressure management (very much intertwined with dialytic process)
- CKD symptom management
- Draw labwork
- Facilitate IV therapies: medications, blood products, antibiotics, IDPN
- Access to allied health: pharmacist, social work, dietitian

HEMODIALYSIS

Managing electrolytes in HD patients

- We do so you don't need to!
- All patients undergo monthly renal labwork
  - CBC, electrolytes, extended electrolytes, PTH, iron studies
- Timing of labdraw for HD patients is important
  - Difference between pre-dialysis vs. intra-dialysis vs. post-dialysis samples
- If there is labwork drawn at LTC of concern – feel free to contact us!

THANK YOU!

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