



Abnormal Sodium

National Pediatric Nighttime Curriculum

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Learning objectives

After this module learners will be able to:

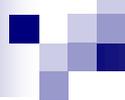
- Describe principles of acute fluid management in the correction of hypernatremia and hyponatremia
- Recognize the signs and symptoms that require immediate attention in patients with disordered sodium
- Consider the level of care appropriate for patients requiring correction of hypernatremia and hyponatremia

Case #1 (intern)

- You have just finished sign out and you are reviewing your patient list to prioritize the most ill patients when your pager goes off:
- “Lab called with critical value for patient in 735: sodium 160. Please advise.
–Kevin”

You review your sign out...

- 7 month old otherwise healthy male admitted directly from clinic in the late afternoon with gastroenteritis and dehydration. He has had minimal PO intake and decreased urine output.
- Tachycardic and febrile when the admitting team saw him but otherwise stable.
- Overnight plan: floor staff is placing an IV, giving a 20cc/kg NS bolus and will call night team to reassess when complete.



You head to room 735

As you go to the bedside to assess the patient, you review some questions:

- What are possible etiologies of hypernatremia?
- What about in this patient specifically?
- What do I need to worry about immediately?
- Should I call my senior?
- Can I take care of this patient on the floor or does he need a higher level of care?

At the bedside

- VS: T 38.5, HR 120, RR 30, BP 90/60, O2 sat 99% RA
- His nurse, Kevin, tells you that the NS bolus is almost complete and that the patient has been irritable since arriving to the floor
- Physical exam: General: irritable infant; HEENT: mucous membranes dry, anterior fontanelle slightly sunken; Chest: clear; CV: tachycardic, regular rhythm, II/VI systolic ejection murmur; Abdomen: soft, hyperactive bowel sounds; Extremities: normal skin turgor, cap refill 3 seconds
- What is your overall assessment of this patient?

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- What is your next step?
 - A) Stop the bolus—this patient is hypernatremic and NS is an inappropriate fluid choice
 - B) Give another 20cc/kg bolus of NS
 - C) Call a renal consult



Next steps

You give another normal saline bolus and the patient's perfusion, heart rate and mental status start to normalize

Kevin asks you what fluids you want to hang now....

- What do you need to consider when correcting the sodium in hypernatremic dehydration?
- What do you need to worry about if correcting too fast?

Calculating free water deficit

- Free water deficit is the minimum amount of fluid necessary to correct serum sodium
- Estimate of free water deficit:
4mL x body weight x desired change in sodium
- Goal is to correct sodium at a rate no faster than 0.5 mEq/L/hour
- Add maintenance fluid needs and account for any ongoing losses



Ongoing management

- What fluid should you choose?
- When should you recheck a sodium?

Hypernatremia

- Defined as serum sodium $\geq 145\text{mEq/L}$

- Causes:

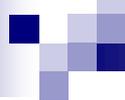
Excess sodium intake	Concentrated formula, salt ingestion (seawater, accidental, Munchausen-by-proxy), hypertonic IV fluids, sodium bicarbonate, blood products
Increased free water losses	<ol style="list-style-type: none">1) Renal: diabetes insipidus, diuretics, tubular disorder2) GI: diarrhea, vomiting, colostomy/ileostomy output, malabsorption3) Insensible: fever, tachypnea, burns
Decreased free water intake	Ineffective breastfeeding, poor access to water, blunted thirst mechanisms, fluid restriction

Clinical Manifestations and Evaluation of Hypernatremia

- Early neurologic signs include agitation and irritability → can progress to seizure and coma
- Neurologic exam can reveal increased tone, brisk reflexes and nuchal rigidity
- Lab evaluation can include:
 - Serum osmolarity
 - Serum glucose
 - Urine osmolarity and specific gravity

Neurologic Sequelae

- In acute phase:
 - Intracellular fluid moves to extracellular space →
volume loss in brain → separation from meninges
- If hypernatremia has existed for >2-3 days:
 - Neurons protect themselves by making osmolytes to maintain gradient
 - With rapid correction, neurons can swell leading to cerebral edema
- Mortality estimated at 10-16% despite correct rate of rehydration



Case #2 (senior)

You are doing your late evening rounds on the ward when one of the nurses pulls you aside:

- “One of the post-op orthopedic patients has a sodium of 115 and I can’t reach the primary team. Can you help me?”

His nurse gives you more info...

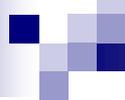
- Patient is a 16yo with cerebral palsy and global developmental delay who is post-operative day #2 from posterior spinal fusion.
- He has been writhing and not tolerating g-tube feeds so has been on maintenance IV fluids of D5 ½NS + 20mEq/L KCl all day.
- His mother is at the bedside and feels he is not himself.

At the bedside

- VS: T 38.0, HR 90, BP 100/75, RR 20, O2 98%RA
- General: neurologically impaired child moaning in bed, less responsive to voice/touch per mother; HEENT: lips dry, mucous membranes slightly dry; Chest: CTAB; CV: RRR, nl S1, S2; Abdomen: g-tube intact, hypoactive bowel sounds; Extremities: well perfused; Neuro: increased tone and spasticity in extremities, responds to voice with a moan, responsive to painful stimuli

Next steps

- You initiate a rapid response and transfer to the PICU should happen shortly. Your immediate next step should be:
 - A) Prompt administration of hypertonic saline (3%)
 - B) Emergent head CT
 - C) Fluid restriction due concern for SIADH



Your patient stabilizes...

Your patient is returning to baseline mental status and you stop the hypertonic saline.

- What general guidelines do you use to think about ongoing fluid management?
- Why are you worried about the rate of correction?

Hyponatremia

- Defined as serum sodium ≤ 135
- Occurs in 3% of hospitalized patients
- Kidney protects against hyponatremia by excreting free water as dilute urine
 - Hyponatremia is an increase in total body water rather than a decrease in serum sodium

Causes of hyponatremia

Decreased total body water	GI losses (diarrhea, emesis), diuretics, RTA, 3 rd spacing
Increased total body water	CHF, acute renal failure, SIADH, water intoxication (dilute formula feeding)
Normal total body water	Hypoglycemia
Pseudohyponatremia	Severe hyperlipidemia or hypoproteinemia

- Hyperglycemia leads to hyperosmolarity with translocation of fluids from intracellular to extracellular space
- Pseudohyponatremia: displacement of plasma water resulting in falsely low serum by laboratory measurement

Clinical manifestations of hyponatremia

- Neurologic symptoms related to edema caused by hypo-osmolarity
 - Children at higher risk due to higher brain-to-skull ratio
- Symptoms include headache, nausea, emesis, weakness
- Severity worsens as edema increases leading to signs of cerebral herniation
 - Respiratory changes, posturing, pupillary changes, seizure

Lab evaluation of hyponatremia

- Serum osmolarity if concerned for pseudohyponatremia
- Urine osmolarity to evaluate for impaired ability to excrete free water
- Urine sodium
 - <25 mEq/L consistent with volume depletion
 - >25 Meq/L consistent with renal tubular dysfunction, SIADH, diuretic use
 - Must be interpreted with caution since affected by IV fluids, fluid restriction, diuretic use

Fluid management goals

- Hyponatremia with neurologic symptoms is a **medical emergency**

Clinical picture	Fluid	Rate
Seizure	3% hypertonic saline	raise serum sodium by 4-8 mEq/L/hour until seizure activity stops
No seizure activity but not at neurologic baseline	3% hypertonic saline	raise serum sodium by 1mEq/L/hour until: -patient at baseline -plasma sodium increases by 20-25mEq/L <u>OR</u> -serum sodium increases to 125-130mEq/L
Asymptomatic	0.9% normal saline	raise sodium no faster than 0.5 mEq/L/hour

Why are we concerned about the rate of correction?

- Excessive changes in serum sodium can lead to cerebral demyelination (central pontine myelinolysis)
 - Usually occurs several days after correction
 - Presents with confusion, quadriplegia, confusion or pseudocoma
- Recent data shows rate of correction may have little affect on development of demyelination
 - Magnitude of correction and underlying illness more important contributing factors
- Risk of untreated hyponatremia far exceeds that of rapid correction so **do not hesitate to use hypertonic saline for symptomatic patients**



Key learning points

- Always prioritize hemodynamic stability over sodium correction
- Correction calculations for both hypernatremia and hyponatremia are general guidelines—sodium should be monitored frequently to ensure safe rate of correction
- Symptomatic hyponatremia is a medical emergency and should be managed in a closely monitored setting with 3% hypertonic saline

References

- Chung C, Zimmerman D. Hyponatremia and hypernatremia: current understanding and management. *Clin Ped Emerg Med*. 2009; 10: 272-278.
- Moritz M and Ayus JC. Disorders of water metabolism in children: hyponatremia and hypernatremia. *Pediatr Rev*. 2002; 23: 371-380.
- Schwaderer AL, Schwartz GJ. Treating hypernatremic dehydration. *Pediatr Rev*. 2005; 26: 148-150.
- Waseem M, Hussain A. Index of suspicion. *Pediatr Rev*. 2004; 25: 397-399.