Approaches to Managing Hypernatremia/Hyponatremia

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

Hyponatremia Facts and Figures

- Hyponatremia is the most common fluid and electrolyte disorder encountered in clinical practice and is found in approximately 20% of admissions to hospital.
- In addition to being extremely prevalent, hyponatremia is an independent predictor of mortality.
- There is a clear, linear relationship between serum sodium <135 mmol/L at the time of admission to hospital and in hospital mortality.
- Efforts to reverse hyponatremia can also be dangerous for the patient.
- Under correction of serum sodium can lead to the development of cerebral edema whereas rapid over-correction of serum sodium can put patients at risk for Osmotic Demyelination Syndrome (ODS)- formerly known as Central Pontine Myelinolysis.

Hyponatremia Facts

- There are two factors which influence how symptomatic a patient will be from their hyponatremia:
  - severity of hyponatremia
  - the acuity of onset.
- The lower the sodium and the faster the fall, the more symptomatic a patient will become.
- Symptoms are often vague and non-specific presenting as headache, irritability, lethargy, confusion, agitation or unstable gait leading to a fall.

Step-Wise Approach to Managing Hyponatremia

- Treat neurologic emergencies related to hyponatremia
- In the event of a seizure, coma or suspected cerebral herniation as a result of hyponatremia, IV 3% hypertonic saline should be administered as soon as possible according to the following guide:
  - Administer 3% hypertonic saline 100-150cc IV over 5-10min
  - Reassess patient
  - If the patient does not improve clinically after the first bolus, repeat a second bolus of hypertonic saline.
  - STOP ALL FLUIDS after the second bolus of hypertonic saline to avoid raising the serum sodium any further
- What if hypertonic saline is not readily available?
  - Administer one amp of Sodium Bicarbonate over 5min.

Step-Wise Approach to Managing Hyponatremia

- Defend the Intravascular Volume
- In order to defend the intravascular volume a determination an assessment of the patient's volume status must occur. Is the patient hypovolemic, euvolemic or hypervolemic?
- Although volume status is difficult to assess with any accuracy at the bedside, a clinical assessment with attention to the patient's history, heart rate, blood pressure, JVP, the presence of pedal and sacral edema, the presence of a postural drop and point-of-care ultrasound (POCUS) is usually adequate to make a rough determination of whether the patient is significantly hypovolemic (requiring fluid resuscitation) or significantly hypervolemic (requiring fluid restriction +/- diuretics).

Hypovolemic Hyponatremia

- In a patient who is hypovolemic and hyponatremic, the priority is to restore adequate circulating volume. In particular, restoring adequate circulating volume takes priority over any concerns that the hyponatremia might be corrected too rapidly and lead to ODS.
- When selecting the type of fluid for restoration of adequate circulating volume in the hypovolemic/hyponatremic patient, be mindful of the sodium concentration of the fluid that you have chosen. Lactated Ringer's has a sodium concentration of 128mmol/L which will be more **ISOTONIC** to the hyponatremic patient.
- Administering Lactated Ringer's will likely result in a slower rise in serum sodium compared to Normal Saline, having a lower risk of potentiating ODS.
- Therefore, Lactated Ringer's is recommended as the fluid of choice for resuscitation of the **hypovolemic/hyponatremic** patient.

Hypervolemic Hyponatremia

- The management of **hypervolemic hyponatremia** centers on sodium restriction, water restriction and diuretics.

Euvolemic Hyponatremia

- Euvolemic patients with hyponatremia have an appropriate volume status, and so no not require any particular treatment to defend intravascular volume, and management should concentrate on preventing worsening hyponatremia.

Preventing Worsening Hyponatremia

- Once you have correctly identified and managed the baseline volume status of the patient through either volume resuscitation or diuresis, the goal becomes preventing further exacerbation of the hyponatremia.
- This is achieved through strict fluid restriction and saline locking the IV. It is extremely important to communicate this to the patient, family and healthcare team. Water can literally kill your patient!

Prevent Rapid Overcorrection: The Rule of 100s

- It is important to understand that the fluid itself that you have given to your patient is not the cause of a rapid increase in the serum sodium, but rather, the free water diuresis that results shortly afterwards.
- Monitoring the urine output will be the deciding factor in preventing overcorrection and possible complications.
- Therefore, to prevent rapid overcorrection:
  - Insert a foley catheter and monitor ins and outs
  - If urine output >100cc/hour, send STAT urine Osmolarity and sodium
  - If urine osmolarity<100, consider 1mcg DDAVP IV
  - Continue following steps 2-4 as per urine output

Indications for DDAVP in patient's with hyponatremia

- Experts recommend administering IV DDAVP in the ED to prevent ODS in the hypovolemic patient with hyponatremia who has been given IV crystalloid for volume replacement and now has a high urine output >100cc/hr and urine osmo <100mosm/L.

Correcting Hyponatremia: The Rule of 6's

- *"Six in six hours for severe symptoms, then stop. Six a day makes sense for safety."*
- The rule of 6s can be helpful in guiding your correction of
- hyponatremia.
- "Six in six hours for severe symptoms and then stop" implies that if you need to rapidly increase serum sodium due to a neurologic emergency do not correct more than 6mmol.
- "Six a day makes sense for safety" implies that you should not exceed an increase of sodium of more than 6mmol/day. While different sources will cite different ranges, targeting six is a conservative approach. If you overshoot by one or two mmol then you will still be well within the safe range.

Ascertain the Cause of Hyponatremia

- Look at Chief complaint: look for conditions which can increase output or decrease intake such as vomiting and diarrhea, pain or altered level of awareness
- Review Medication List: look for those that cause SIADH, especially thiazide diuretics and SSRIs; patients who have been on chronic steroids may have adrenal insufficiency
- Evaluate PMHx: Look for history of end organ failure (CHF, liver failure and renal failure) or cancers (a common cause of SIADH)
- Lab work: hyperglycemia, hyperkalemia (may suggest adrenal insufficiency), hypothyroidism
- **Pearl:** Correcting hypokalemia can help improve hyponatremia

Complications of Hyponatremia

- Cerebral Edema
  - Cerebral edema should be considered in all patients with either severe hyponatremia or a rapid lowering of serum sodium concentration and altered level of consciousness.
  - Measurement of the optic nerve diameter can be done with point-of-care ultrasound (POCUS), and a CT scan of the head may show effacement of the sulci as a surrogate of cerebral edema.
  - If you suspect cerebral edema, administer 3% hypertonic saline as described.

Complications of Hyponatremia

- Osmotic Demylination Syndrome (ODS)
  - Formerly known as Central Pontine Myelinolysis, ODS is a devastating condition which can occur after rapid overcorrection of hyponatremia.
  - It is a clinical diagnosis with a delayed presentation up to 7 days after the rapid correction. The symptoms can vary and are dependent on which anatomical structure in the brain demyelinates.
  - ODS most commonly affects the pons, however other structures can be affected including the cerebellum or basal ganglia.
  - Commonly described symptoms include ataxia, quadriplegia, cranial nerve palsies, and the 'locked-in' syndrome.
- Risk factors for ODS:
  - elderly
  - malnourished state
  - chronic severe hyponatremia
  - hypokalemia

Management of the patient who has been overcorrected

- The scenario we all dread seeing in a hyponatremic patient occurs when a repeat serum sodium level comes back dramatically higher than expected. The management of overcorrection of hyponatremia is similar to the general approach to hyponatremia:
  - Defend the intravascular volume
  - Prevent the sodium from increasing any further
    - Fluid restriction: make the patient NPO and stop IV fluids
    - Give DDAVP 1mcg IV
    - Consult Nephrology

Exercise Associated Hyponatremia (EAH)

- EAH is most commonly seen among endurance athletes.
- Their hyponatremia is a result of ingesting more free water than they are able to clear by voiding which leads to an overall excess of free water as the kidneys are not able to excrete it.
- The cognitive trap is to assume that their symptoms (and electrolyte abnormalities) are a result of dehydration given their exercise history.
- Don’t assume that DEHYDRATION is the cause.
- The real culprit is an excess of free water.
- Therefore, the treatment of EAH is fluid restriction!

Psychogenic Polydipsia

- Psychogenic polydipsia is typically seen in psychotic patients who ingest large quantities of free water.
- Patient's who are concurrently taking SSRIs (which may cause SIADH) are especially at risk of developing severe hyponatremia.

Hypernatremia Facts and Figures

- Hypernatremia is defined as a serum sodium value > 145 mEq/L. Since the serum sodium is determined by the ratio of the amount of sodium in the serum to the amount of plasma water, hypernatremia can develop from either an excess of sodium (such as due to the administration of hypertonic fluids), a loss of hypotonic fluids (free water) or a combination of both.
- Most commonly, it is the loss of hypotonic fluids and the failure to replace these water losses that result in hypernatremia.
- In most circumstances, thirst is a powerful defense mechanism against a rise in the serum sodium level. The body defends its serum osmolality closely so that as the serum sodium rises (and with it serum osmolality), thirst ensues along with rises in arginine vasopressin (AVP) secreted by the posterior pituitary.
- AVP leads to urinary concentration and conservation of renal water excretion, but ultimately it is thirst and ingestion of water that allows the serum sodium to normalize. Thus, most patients who develop hypernatremia have the common feature that water intake is restricted in some form.

Hypernatremia Facts and Figures

- Given the powerful ability of thirst to defend against hypernatremia, it is not surprising that the incidence of this electrolyte disorder in patients presenting to the emergency department (ED) is uncommon (0.2%).
- Most of these patients usually have either chronic or acute impairment in their mental status (such as dementia). In critically ill patients, the incidence of hypernatremia is 10-fold higher (2-6%).Importantly, a large percentage of patients develop hypernatremia during the course of their hospital stays (especially in the ICU – up to 10%).
- The reasons for this are multi-fold and include:
  - Use of hypertonic fluids
  - Ongoing loss of body fluid loss (gastric decompression, stool, fistulas, biliary drains, etc., inattention to water intake and needs)
  - Use of diuretics, lactulose
  - Poorly controlled hyperglycemia.

Hypernatremia Facts and Figures

- The clinical symptoms associated with hypernatremia are alterations in central nervous system functioning, including a spectrum ranging from mild confusion to stupor and coma.
- These symptoms likely result from changes in cellular volume as water moves from the intracellular compartment to the more hypertonic extracellular compartment resulting in cell shrinkage.
- Mortality rates in patients with hypernatremia, especially those in the ICU, are very high (ranging from 15 to 50%), depending upon the severity of the hypernatremia.
- While hypernatremia has an independent effect on increased mortality, the underlying disease processes driving the development of hypernatremia is more likely to blame with the higher mortality rates.

Causes of Hypernatremia

- Water with solute loss (with water losses in excess of solute losses)
  - Cases of water with solute loss and pure water losses, most patients will have impaired mental status and decreased thirst sensation, or the inability to obtain free water
- Pure water losses
- Solute (Sodium) gain

Causes of Hypernatremia

- Extra-renal water losses (profuse sweating)
- Renal losses (osmotic diuresis)
- High-protein intake
- Lithium administration
- Vasopressin receptor antagonists
- High urine output that is hypotonic to plasma.
- Lastly, solute gain occurs in situations where hypertonic solutions are given to the patient (for example, sodium bicarbonate during a cardiac resuscitation).

At Risk Population's for Hypernatremia

- Disturbed sensation of thirst
- Unconscious
- Water intake dependent on another clinician/individual o Immobile patient
- Altered mental status
- Enterally fed patients
- Critically ill patients

Correction of Hypernatremia

- As many patients with hypernatremia will be volume depleted as well as dehydrated, assessing the need for rapid resuscitation is critical, and if needed, intravenous isotonic solutions should be administered until the patient is hemodynamically stable.
- Before correction of hypernatremia, it is vital to determine if the rise in serum sodium is acute (< 48 hours) or more chronic (> 48 hours). This is because with chronic hypernatremia, brain adaptations in cellular volume have occurred such that rapid correction in these circumstances can result in cerebral edema, increased intracranial pressure and brain stem herniation with death.
- In chronic states of hypernatremia the serum sodium should not be lowered by more than 8 to 10 mmol/L/24 hours. If it is unclear as to the duration of hypernatremia, it is best to assume that the condition is chronic and use a slower rate of correction.

Correction of Hypernatremia

- Several formulas are available to determine the rates of infusion of hypotonic solutions and any of these can be utilized.
- However, it is critical that the clinician measure serum sodium levels frequently the duration of correction (every 4-6 hours), so that over-rapid correction is avoided and infusion/replacement rates for water can be adjusted.
- Water replacement can be achieved in several manners:
  - Intravenous hypotonic fluids which may range from 0.45% saline to 5% dextrose in water
  - Enteral water administration.

Oral vs. IV Hydration

- If you are contemplating oral hydration, it is important to assess the ability of the GI tract to accommodate and absorb the additional fluid load.
- This is especially true if the patient is already receiving EN and the additional volume with hydration may prove intolerable.
- Certainly, if a patient is having trouble tolerating EN, then IV hydration should be the route of choice.
- Other key questions that need to be asked before utilizing enteral hydration include:
  - Will enteral solutions be held for periods of time (such as for procedures, lost access)?
  - Does the patient have impaired GI motility?
  - Is the patient constipated?
  - Is the patient at risk for aspiration that could be worsened by increased GI distention?

Oral vs. IV hydration

- Finally, it should be noted that when large and frequent water flushes are ordered (such as 300mL every 4 hours), each time the flush is scheduled to run in, enteral feedings are stopped or automatically shut off (if the patient is on a dual pump), while the water infuses.
- The larger the flush, the more time it takes to run in. If the patient has frequent and large flushes, this can translate into significant lost feeding time and the patient's nutritional status is compromised.
- Furthermore, if the above dual enteral pump is being used and the patient is on a nocturnal, or other cycled delivery mode, when the pump is turned off, no flushes will be infused.

Suggested Guidelines

- For serum sodium < 150 mmol/L, it is reasonable to try enteral water replacement up to 1 liter in divided doses (for example, 250mL every 6 hours, or 165mL every 4 hours).
- For serum sodium > 150 mmol/L, IV hydration should be given carefully, and in a controlled and reliable fashion, using dextrose 5% in water or another hypotonic fluid as appropriate for the individual patient.