# EM CASES SUMMARY

## Episode 60 – Emergency Management of Hyponatremia

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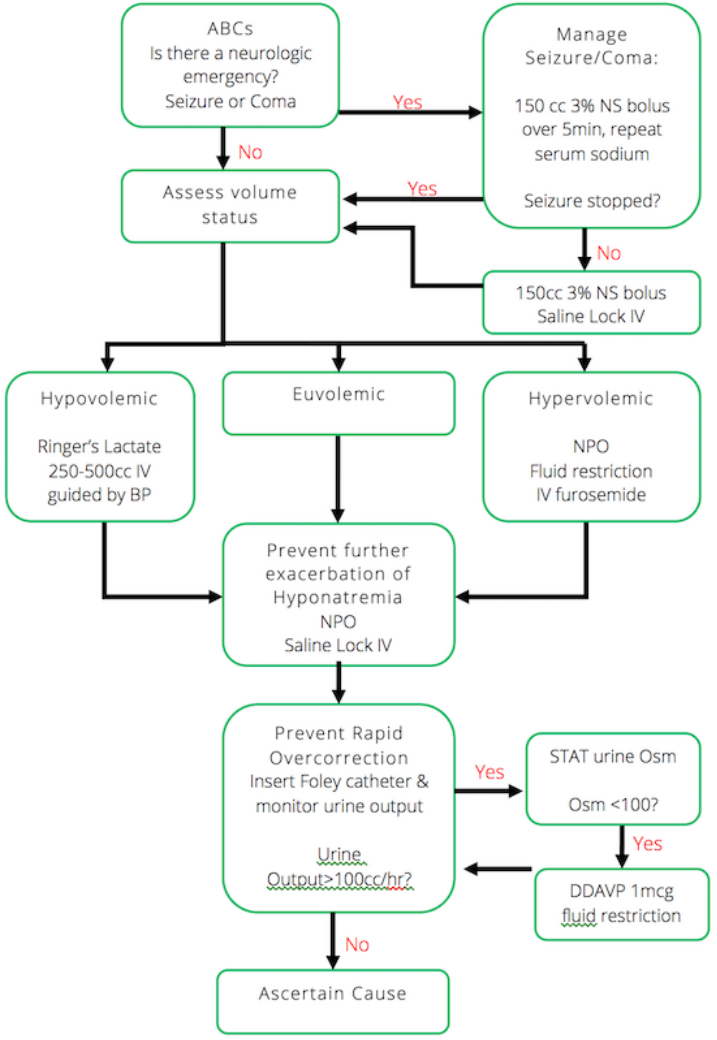
Hyponatremia is the most common fluid and electrolyte disorder encountered in clinical practice and is found in approximately 20% of admissions to hospital. In addition to being extremely prevalent, hyponatremia is an independent predictor of MORTALITY. There is a clear, linear relationship between serum 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.

There are two factors which influence how symptomatic a patient will be from their hyponatremia: severity of hyponatremia and 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.

## Approach to Hyponatremia

*Conceived by Dr. Edward Etchells*



# Step-wise Approach to Managing Hyponatremia

## 1. 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:

1. Administer 3% hypertonic saline 100-150cc IV over 5-10min
2. Reassess patient
3. If the patient does not improve clinically after the first bolus, repeat a second bolus of hypertonic saline.
4. 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.

## 2.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 (helpful in Dr. Etchell's opinion) 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).

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 osmotic demyelination syndrome.

When selecting the type of fluid for restortation of adequate circulating volume in the hypovolemic/hyponatrmemic patient, be mindful of the sodium concentration of the fluid that you have chosen. *Ringer's lactate* has a sodium concentration of 128mmol/L which will be more ISOTONIC to the hyponatremic patient. Administering *Ringer's lactate* will likely result is a slower rise in serum serum sodium than Normal Saline, and therefore have a lower risk of potentiating osmotic demyelination syndrome. *Ringer's lactate* is therefore recommended by our experts as the fluid of choice for resuscitation of the hypovolemic/hyponatremic patient.

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

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.

## 3. Prevent 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!

## 4. 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:

1. Insert a foley catheter and monitor ins and outs
2. If urine output >100cc/hour, send STAT urine Osmolarity and sodium
3. If urine osmolarity<100, consider 1mcg DDAVP IV
4. Continue following steps 2-4 as per urine output

### Indication for DDAVP in patients with hyponatremia

The situation in which our experts recommend administering IV DDAVP in the ED to prevent osmotic demyelination syndrome is in the hypovolemic patient with hyponatremia who has been given IV chrystalloid for volume replacement and now has a high urine output >100cc/hr and urine osmolarity <100mosm/L.

### Correcting Hyponatremia: The Rule of 6s

*"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 make 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.

## 5. Ascertain the Cause of Hyponatremia

1. 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
2. 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
3. Evaluate PMHx: Look for history of end organ failure (CHF, liver failure and renal failure) or cancers (a common cause of SIADH)

4. Lab work: glucose (hyperglycemia), potassium (hyperkalemia may suggest adrenal insufficiency), TSH (hypothyroidism)

Pearl: Correcting hypokalemia can help improve hyponatremia

Options

- 1 tab of 20meq of KDur tid (preferred by our experts)
- KCl elixir 10mmol q4H PO or NG
- Ringers lactate 50mL/hr IV + 40KCl/hr

## 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 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.

Video on how to perform ocular ultrasound for optic nerve diameter: https://vimeo.com/41575053

If you suspect cerebral edema, administer 3% hypertonic saline as described.

### 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:

1. elderly
2. malnourished state
3. chronic severe hyponatremia
4. 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:

1. Defend the intravascular volume
2. Prevent the sodium from increasing any further
    a. Fluid restriction: make the patient NPO and stop IV fluids

b. Give DDAVP 1mcg IV

3. 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. This 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 EHA is fluid restriction!

## Psychogenic Polydypsia

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.

## Key References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