Hyponatremia – Dr Mok Shao Feng

Reference

EJE CPG on diagnosis and treatment of hyponatremia: https://eje.bioscientifica.com/view/journals/eje/170/3/G1.xml

When to give 3% hypertonic saline? 0:54

- Indication: Severe hyponatremia (<120) in the context of neurological compromise (e.g. confusion/delirium/stupor) regardless of etiology
- CAVEAT: Bear in mind that onset of symptoms is due to cerebral edema. In the context of acute hyponatremia (less than 48 hours), patient can still develop symptoms if Na acute drops from normal to 120s as brain has no time to compensate
- This also explains why some patients can walk about and are asymptomatic with a Na <120mmol/L. This indicates chronic hyponatremia (> 48 hours) and brain cells lose salts to reduce intra-cellular osmolality and guard against cerebral edema.
- Exclude other causes of delirium especially if sodium levels not too low (e.g > 125mmol/L). Bear in mind that this is an arbitrary cut-off.

How to give 3% hypertonic saline? 3:30

- Care should be escalated to endocrine consultant or registrar on call
- Aim is to bring sodium up by **4-5 mmol** with the intention of relieving cerebral edema; beyond which there is risk for osmotic demyelination
- Dosing:
  - 2ml/kg up to 150ml as single bolus (each bolus increases Na by 1-2mmol)
  - Can give over 30-60 min
- Recheck sodium 30-60 min after hypertonic saline given
  -> Can repeat and give 1 more 3% fluid bolus (2ml/kg) if symptoms are not improving. No more than 2 boluses
- Symptomatic improvement might be delayed
- If sodium improves but mental status does not, consider alternative causes of delirium

Which patients require monitored care? 6:58

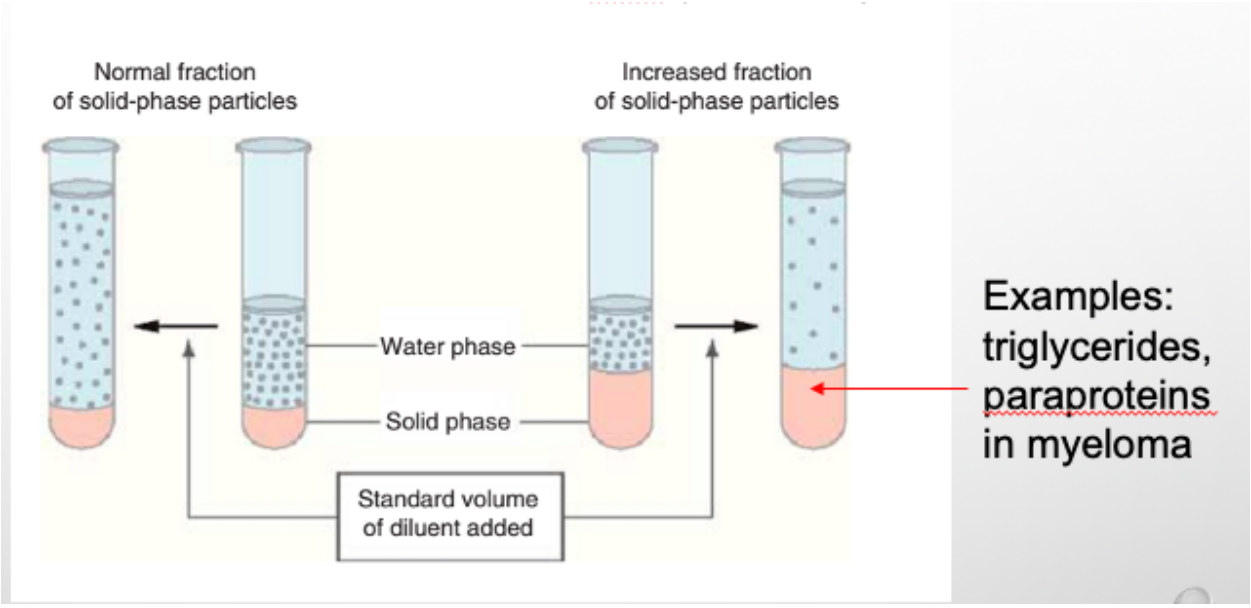
- When patients lose ability to protect airway
- Although, can consider monitored care if 3% hypertonic saline is required

Calculated vs measured serum osmolality? 8:30

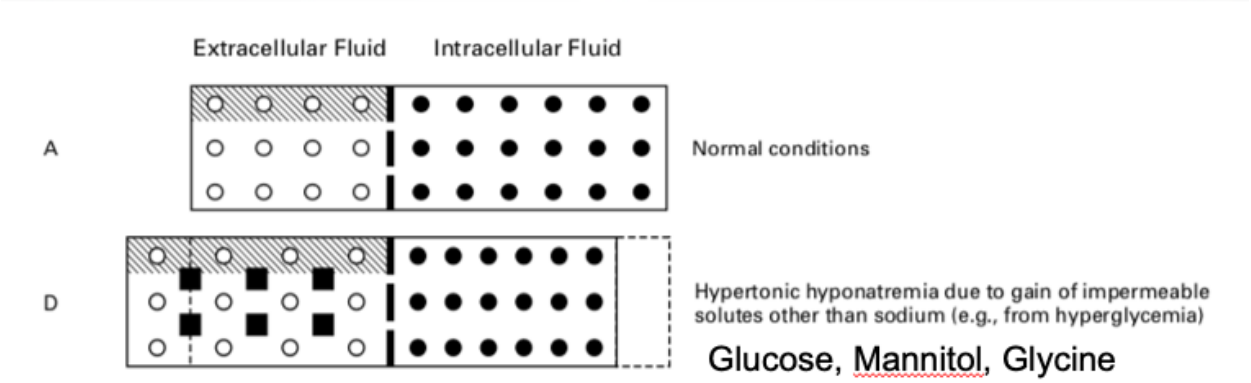
- **Most hyponatremias are hypo-osmolar because sodium is the key contributor to osmolality**
- Most instances, calculated and measured osm should be quite close – hence usually just use measured osmolality
- In hyperglycemia, will need to check corrected sodium to determine Na level when patient becomes euglycemic

Causes of Non-Hypo-Osmolar hyponatremia 12:08

- Pseudohyponatremia
  - Causes: Hypertriglyceridemia, parapoteinemia (myeloma)
  - Mechanism: Increase solid fraction in test tube results in relatively increased dilution of sodium when diluent added
  - Test on iSTAT machine to overcome this issue
- Hyperosmolar Causes
  - Causes: Commonest is hyperglycemia (others: mannitol, glycine)
  - Glucose pulls waters from ICF into ECF diluting out sodium
  - This is TRUE hyponatremia (not pseudohyponatremia) where salt is diluted in vivo



Pseudo-hyponatremia



True hyponatremia (often hyper-osmlar) related to other solutes exerting osmotic effect and pulling water from ICF into ECF.

Urinary Studies 16:24

- When to send urinary studies?
  - **Often not needed in patients who are in frank fluid overload**
  - Usually sent in patients who are dehydrated vs SIADH/hypocortisolism/hypothyroidism
- **All patients with sodium < 125 should be evaluated** – should not attribute to dehydration
- Dehydration related hyponatremia is overcalled; extent of dehydration has to be quite severe to cause hyponatremia, SIADH is in fact more common
- Timing of sending studies:
  - Generally, the earlier the better
  - However, if patients are on diuretics, might be difficult to interpret urinary sodium
- If patient is on diuretics
  - If urine sodium is low despite being on diuretics, quite definitely volume depleted
  - But if urine sodium is high, then might be difficult to interpret; might consider holding off and repeating
  - Can use uric acid and urea and FENA as adjuncts to determine if patient is dehydrated

What do urinary sodium and osmolality mean? 23:44

- Urine osmolality
  - When serum osm is < 280, body should be producing close to no ADH, and urine should be maximally diluted at <100 mOsm/kg H2O
  - Hence if urine osm >100 mOsm/kg H2O, this implies dysregulated ADH action
- Urine Na
  - Decrease effective circulating volume (dehydration, nephrotic syndrome, heart failure, cirrhosis): Urinary sodium < 30mmol/L because aldosterone maximally re-absorbing Na at DCT
  - SIADH/Hypocortisolism/hypothyroidism/thiazide diuretics: Urinary sodium > 30mmol/L

Dehydration + SIADH – What to do? 29:25

- If initial urinary studies suggest dehydration but sodium starts to decrease with IV hydration, may consider repeating urinary studies as patient may have underlying concomitant SIADH
- **Caution with blindly giving isotonic saline when patient has hyponatremia (especially when sodium levels are low) – isotonic saline can worsen hyponatremia**
- Hyponatremia due to volume contraction is not common, SIADH is commoner in inpatient setting
- If patient is hemodynamically stable, and there is no pressing need for IV hydration, advisable to work up before blindly hydrasting

Underlying kidney disease/cirrhosis? 33:26

- In advanced cirrhosis or CKD, hyponatremia is usually attributed to underlying disease
- However in milder CKD, can consider performing urinary studies as discussed above

SIADH? 37:00

- SIADH is a diagnosis of exclusion – **exclude hypocortisolism, hypothyroidism, thiazide diuretic use**
- Significant proportion are idiopathic; possible senescence underpinning
- **Common etiologies: Chest infections, sepsis, paraneoplastic, pain, trauma, nausea, drugs (SSRIs, TCAs, chemo drugs)**
- Take a good history for malignancy related symptoms
- Subjective threshold for brain imaging

Monitoring Frequencies and Troubleshooting 41:11

- Based on mental status, sodium levels and trajectory, daily weight and urine output
- Frequency of serum sodium checks: Severe hyponatremia usually start with 4-6 hourly initially, once more stable then can gradually space out
- Rate of correction: Aim 6-8mmol rise every 24 hours to minimise risk of osmotic demyelination as a complication
- At risk groups:
  - **Patients with hypocortisolism who are treated with steroids are at risk for rapid rise in serum sodium**
  - If urine output > 100ml/hour – that is when serum sodium will likely surge and when help needs to be sought
- Modulating rate of rise of sodium when rise is too rapid
  - Escalate to endocrine if the need arises
  - Options: Hypotonic/isotonic fluids (1-1.5L/24 hours), DDAVP – to be given judiciously AND ONLY UNDER SUPERVISION OF ENDOCRINOLOGY TEAM
  - Will require close monitoring
- Take home: Escalate when sodium rise > 6-8 mmol/24 hours or when patients urine output > 100ml/hr

Treatment for SIADH 47:05

- Fluid restriction: Depends on 1) urine osm 2) how much osmolytes are consumed
  - The higher the urine osm, the stricter the fluid restriction must be

- Increasing osmolyte consumption to facilitate free water expulsion
    - High protein diet – broken down into urea which binds to water and gets cleared
    - If sodium is still not improving despite fluid restriction, can consider sodium tablets
        - § Start low e.g. 1 tablet BD, go slow, slowly uptitrate
        - § Sodium chloride tablets should be reviewed and not continued on it indefinitely
- If trigger for SIADH has resolved, generally don't need fluid restriction

Take Home Points

- Hyponatremia is usually a problem of free water excess, no sodium deficit
- Establish whether hyponatremia is severe, and escalate and commence hypertonic saline treatment
- Etiology:
    - Most hyponatremias are hypoosmolar
    - Dehydration is not as common as you think
    - SIADH is more common than you think but it is a diagnosis of exclusion

- Always investigate if Na < 125 – don't just attribute to dehydration
- Don't give fluids unnecessarily pre work up especially if sodium is low, fluids can cause harm by further lowering sodium in SIADH due to the saline paradox (See below). So long as patient is hemodynamically stable, it is always possible to evaluate etiology hyponatremia before deciding if patient will benefit from fluid restriction or IV fluids.