ELECTROLYTE ABNORMALITIES

SODIUM

Hyponatremia

Causes:

Plasma osmolarity

Isotonic
hyperlipidaemia
paraproteinaemia

Hypotonic
mannitol

Hypertonic
glucose

Extrarenal loss:

Water overload:

cardiac failure
cirrhosis
nephrotic syn
renal failure

hypovolaemia

hypervolaemia

Euvolaemia

Water overload:

stress
drugs
renal insufficiency
diuretics
mineralocorticoid deficiency
salt losing nephritis
cerebral salt wasting

Clinical Features:

Nausea, vomiting, headache, confusion, seizures and coma

Treatment:

Depends on underlying cause. (e.g. fluid restriction in SIADH)

Rapid correction is only indicated in symptomatic patients (e.g. convulsions)

100mmol - 250mmol Na over 10min

after which the rate of correction must be slow
Acute (<3 days)
   No greater than 2mmol/l/hr, do not raise Na >12mmol/24hours
Chronic (>3 days)
   No greater than 0.5mmol/l/hr, do not raise Na >12mmol/24hours

Important:
1. In patients where you are not sure whether hyponatremia has developed acutely or chronically, assume it has developed chronically.
2. Correct Na slowly because of the risk of central pontine myelinolysis.
3. Plasma Na must be monitored closely.
4. If hypertonic saline is to be used, must discuss with ICU senior.

Note the sodium content in the various Na supplement:
- NaCl tablet 600g 10mmol
- NaCl 0.9% 0.15mmol/ml
- NaCl 3% 0.5mmol/ml
- NaCl 23.4% 4mmol/ml
- NaHCO₃ 8.4% 1mmol/ml
Estimated Na deficit = 0.6 x BW x (desired – current Na)

Hyponatraemia
Causes:

- Clinical evaluation
  - check urine/plasma osmolarity
  - >1 extrarenal loss
  - = 1 osmotic diuresis
  - <1 diabetes insipidus

- Hypovolaemia
  - Hypotonic fluid depletion
    - iatrogenic
    - mineralocorticoid xs

- Euvolaemia
  - Pure water depletion

- Hypervolaemia
  - Salt gain

Note:
Hypotonic fluid depletion = isotonic fluid loss and pure fluid loss
Isotonic fluid loss from extracellular component => minimal fluid shift
  ➞ small increase in sodium and osmolarity
  ➞ but vascular collapse
Pure water depletion
⇒ water loss shared between intra and extracellular components
⇒ minimum circulatory effects
⇒ but large increase in sodium and osmolarity

Clinical features:
Thirst, lethargy, seizures and coma

Treatment:
Depends on underlying cause/ correct underlying problems
(e.g. treat DI with ddAVP)
Correct systemic haemodynamics (isotonic component)
   With 0.9% saline
Correct water deficit (pure water component)
   Water deficit = 0.5 x BW X (plasma Na/140 - 1)
   Replace with water orally, ½ saline, D5 solution
Dialysis may be considered

Important:
1. Correct Na slowly because of possible cerebral oedema
   Max rate = 0.5mmol/l/hour
2. Plasma Na must be monitored closely.
POTASSIUM
Hypokalaemia
Causes: (list not exhaustive)
Drip arm effect
Decrease intake
Increase loss: GI – vomiting, huge gastric aspirate, diarrhea, fistula
    Renal – primary and secondary hyperaldosteronism
        excess mineralocorticoid
    drugs – diuretics, amphotericin, gentamicin
    chronic pyelonephritis, polycystic kidney
    diuretic phase of ATN
    renal tubular acidosis
    magnesium deficiency
Compartmental shift: hypothermia
    alkalosis
    insulin
    refeeding syndrome
    treatment of severe megaloblastic anaemia
    hypokalaemic periodic paralysis
    β2 agonists

Clinical features:
CVS - ECG changes (flattening of T waves, ST depression, U waves, prolong QT)
    Arrhythmia - SVT, VT, torsades
GI - ileus, constipation
CNS – cramps/ parasthesia/ weakness/ tetany/ rhabdomyolysis
Others - glucose intolerance/ renal polyuria/ metabolic alkalosis

Treatment:
Treat underlying cause (e.g. replace Mg/correct alkalosis)
Oral route: syrup KCL – 1G = 13.4mmol
    slow K - 600mg = 8mmol
intravenous: 10 – 20 mmol in 100 ml NS/D5 over 1 hour
    This is a very concentrated K supplement and must be administered
    via central line
Hyperkalaemia
Causes: (list not exhaustive)

Pseudohyperkalaemia
Excessive intake:
- exogenous - K supplement/ massive transfusion
- endogenous - burns, trauma, rhabdomyolysis/ tumor lysis

Decrease renal excretion:
- Addison's disease
- hypoaldosteronism
- drugs - K sparing diuretics, indomethacin etc.
- renal failure

Compartmental shift: acidosis
- Insulin deficiency
- Drugs - suxamethonium
- Hyperkalaemic periodic paralysis

Clinical Features:
CVS - ECG changes (tall T waves, flat P waves, prolonged PR interval, prolonged QRS, sine waves)
- Arrhythmia - VF
- Muscle weakness

Treatment:
- Treat underlying cause
- Urgent situations - can cause cardiac arrest (follow ACLS guidelines)
  - 50 - 100ml 8.4% NaHCO₃ IV
  - 10ml 10% CaCl₂ IV
- enhance K shift to cells:
  - 10 units actrapid + 50ml 50% D50 over 20 min
    (close monitor K and H'stix)
- enhance K excretion:
  - resonium A (sodium polystyrene sulphonate)
  - resonium C (calcium polystyrene sulphonate)
  - oral - 15g Q6H
  - rectal - 30g Q12H
  - consider dialysis
CALCIUM

Hypocalcaemia
Ionized Ca is more physiological important than total calcium
Adjusted Ca = (40 - albumin)/40 + measured Ca

Causes: (list not exhaustive/ many types of classification)
Factitious
Reduction in ionized Ca with normal total plasma calcium
  Respiratory alkalosis
  Citrate toxicity
Decreased PTH activity
  Hypoparathyroidism
  Pseudohypoparathyroidism
  Hypomagnesaemia
Vitamin D deficiency
Others
  Critical illness (sepsis, burns)
  Pancreatitis
  Rhabdomyolysis
  Hyperphosphataemia

Clinical Features:
CVS – hypotension
  bradycardia
  insensitivity to catecholamine and digoxin
  ECG changes – QT/ST prolongation
Neuromuscular – anxiety, psychosis, confusion, seizures
  Tetany, cramps, parasthesia
  Laryngospasm, bronchospasm

Treatment:
Emergency - bolus 2.5-5mmol over 10min
Intravenous - Ca gluconate 10ml = 2.3mmol
  Ca chloride 10ml = 6.8mmol
Oral - Ca carbonate (Oscal 500mg/tablet, caltrate 600mg/tablet)
  Usual requirement 1000mg/day
Measure ionized Ca levels
Most experts do not routinely recommend treating ionized Ca levels unless
<0.8mmol/L or if symptoms develop.
Hypercalcaemia
Causes: (common causes)
Hyperparathyroidism
Malignancy
Granulomatous disease
Immobilization
Thyrotoxicosis
Excess vitamin D intake

Clinical features:
CVS – hypertension
    arrhythmia
    ECG changes – QT shortening
GI - anorexia, constipation
    peptic ulcer, pancreatitis
CNS – depression, retardation, coma, seizure
    Weakness, areflexia, atrophy
Others skeletal problems
    Renal problems e.g. nephrocalcinosis, tubular dysfunction, DI

Treatment:
1. general measures
   • remove offending cause. Treat underlying cause.
   • dietary restriction
   • hydration (dilution effect)
2. increase Ca excretion
   • saline 2-3L over 3-6 hours
     maintain urine output 200ml/hour
     frusemide 10-40mg Q4H
   • consider dialysis
3. decrease bone resorption (should only be given after specialist advice)
   calcitonin – onset 6-10hours
   glucocorticoids
   biphosphonate, mithramycin
MAGNESIUM

Hypomagnesaemia
Causes: (list not exhaustive)
- GI loss
  - Reduced intake
  - Reduced absorption
  - Vomiting, prolonged diarrhea, refeeding syndrome
- Renal loss
- Drug induced loss – e.g. diuretics, amphotericin, aminoglycoside
- Others – burns, sepsis, cardiopulmonary bypass, Mg free dialysate

Clinical Features:
- CVS – arrhythmia, coronary artery spasm, CHF, prolong PR and QT
- GI - anorexia, dysphagia, cramps
- CNS - muscle weakness, lethargy, seizure, confusion, irritability
- Associated with hypokalaemia and hypocalcaemia

Treatment:
- Severe symptomatic hypomagnesaemia – 10mmol MgSO₄ over 5 min
- Asymptomatic - 10mmol MgSO₄ over 2 hours
- Preparation MgSO₄ 1g= 98mg Mg = 4mmol
- Cautious replacement in patients with renal failure

Hypermagnesaemia
Causes: usually iatrogenic

Clinical Features:

<table>
<thead>
<tr>
<th>Plasma concentration (mmol/L)</th>
<th>Effects</th>
</tr>
</thead>
<tbody>
<tr>
<td>0.7 – 1.0</td>
<td>normal range</td>
</tr>
<tr>
<td>2.0 – 3.0</td>
<td>therapeutic range</td>
</tr>
<tr>
<td>3.0 – 3.5</td>
<td>ECG changes</td>
</tr>
<tr>
<td>4.0 – 6.0</td>
<td>areflexia</td>
</tr>
<tr>
<td>6.0 – 7.0</td>
<td>respiratory arrest</td>
</tr>
<tr>
<td>10.0 – 12.5</td>
<td>cardiac arrest</td>
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</tbody>
</table>

Treatment:
- Stop Mg supplement
- NM effects antagonized by 10ml 10% Ca gluconate
- Normal renal function - frusemide
- Renal failure - dialysis may be necessary
**PHOSPHATE**

**Hypophosphataemia**

**Causes:**
- GI - decrease oral intake
  - malabsorption
  - fistula
  - diarrhoea
- Renal - increase loss
  - diuretics, steroids
  - haemodialysis
- Others - hyperparathyroidism, vitamin D deficiency,
  - alcoholism, treatment of DKA, refeeding syndrome,
  - burns, alkalosis

**Clinical Features:**
- CVS - myocardial depression, hypotension and heart failure
- Resp - respiratory failure
- CNS - confusion, delirium, seizures
- GI - anorexia, vomiting
- Renal - renal tubular acidosis, hypermagnesuria, hypercalciuria
- Haemat - haemolysis, platelet and leucocyte dysfunction
* Skeletal - muscle weakness, rhabdomyolysis, difficult to wean

**Treatment:**
Severe hypophosphataemia/symptomatic hypophosphataemia
- KH₂PO₄/K₂HPO₄ 10ml = 14.5mmol PO₄, 18.5mmolH and 25mmol K
- Replace 5 - 10ml in maintenance fluid or normal saline over 6 hours
  - (check K and PO₄ and Ca after replacement)
  - Consider sodium phosphate if hyperkalaemia

**Oral supplement:** mist PO₄ 10ml Q8H
  - (0.4mmol/ml)

**Note:** [Ca][PO₄] >6
  - Risk of Calcium deposition in soft tissue

**Hyperphosphataemia**

**Causes:**
- Factitious - haemolysis, sample separation delay
- Redistribution - trauma,
  - rhabdomyolysis,
  - acidosis (keto and lactic)
tumor lysis
diphosphonate therapy
Positive phosphate balance - acute phosphate administration
phosphate enema
excess IV administration
vitamin D toxicity
Renal retention - renal failure
hypoparathyroidism
pseudohypoparathyroidism
acromegaly
Clinical Features:
(Not well documented. Symptoms are usually caused by the accompanying hypocalcemia and not the hyperphosphataemia per se)
Others - nephrocalcinosis, nephrolithiasis, band keratopathy
Treatment:
Usually aims at correcting the underlying hypocalcemia.
2 approaches to correct hyperphosphataemia:
1. Promote PO$_4$ binding in the upper GI tract (ie. GI dialysis)
   - lower the serum PO$_4$ even in the absence of any oral intake phosphate
   - sucralfate or aluminium-containing antacids
   - calcium acetate tablets if significant hypocalcemia
     (each calcium acetate tablet = 667 mg contains 8.45 mmol calcium.
      Recommended dose 2 tab tid)
2. Enhance PO$_4$ clearance
   - hemodialysis. Reserved for patients with renal failure